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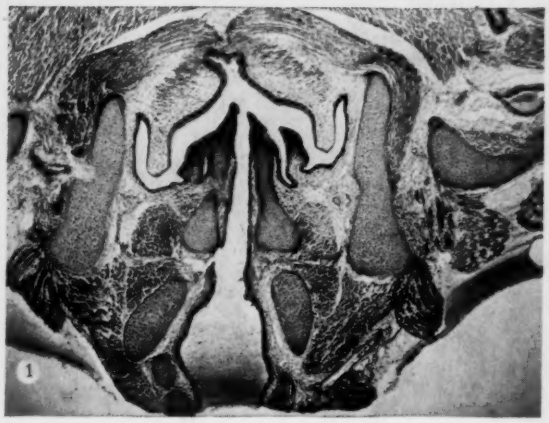

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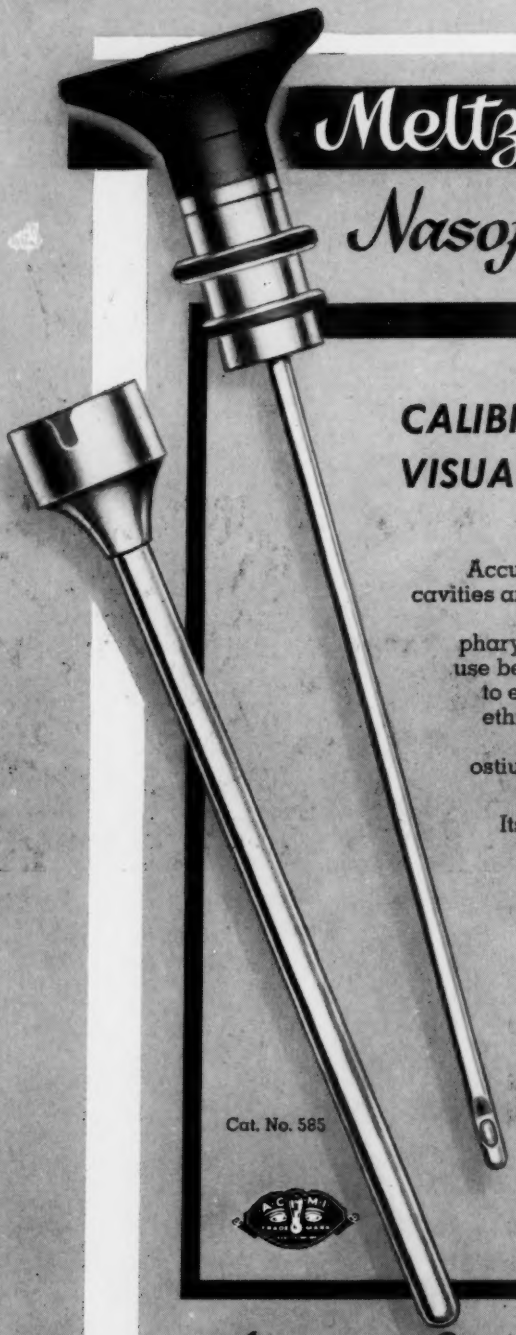

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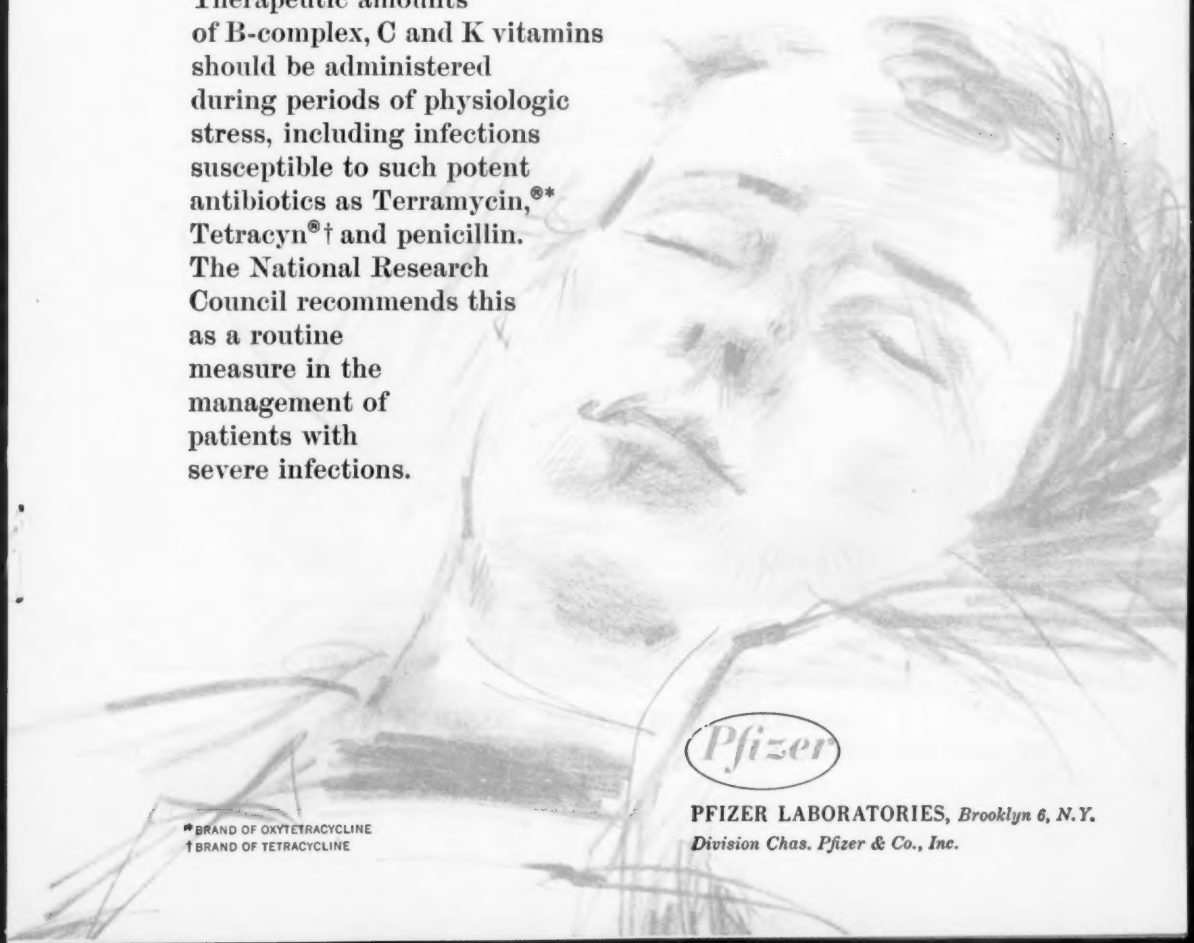

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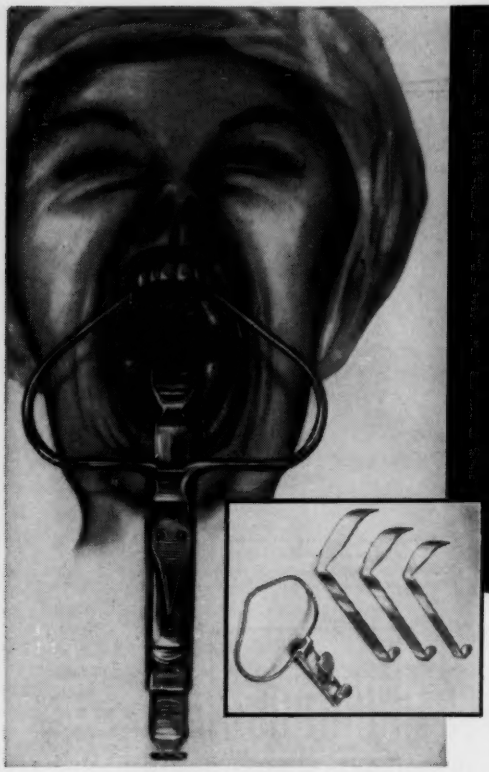

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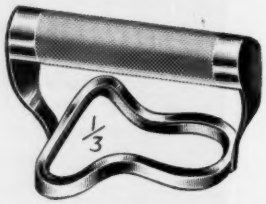

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Borderline Malignant Lesions of the Larynx

F. JOHNSON PUTNEY, M.D., Philadelphia

Borderline malignant lesions of the larynx pose a problem to the physician not only in the selection of treatment but more especially in the diagnosis of their malignant tendencies. It is obvious that if one can detect a premalignant lesion before there is evidence of tissue invasion and metastasis the chances of obtaining a cure are enhanced. However, this diagnosis of premalignancy is extremely difficult both clinically and pathologically. Since we do not know what factors are responsible for malignant degeneration, it cannot be accurately predicted which lesions will become malignant or after what length of time. Certain laryngeal lesions must always be considered as predisposing to malignancy, making it necessary to check them both clinically and histologically over prolonged periods and at frequent intervals. Even so, their management is ofttimes complex and confused. There are four principal laryngeal lesions that should be classified as premalignant: keratosis, carcinoma in situ, the salivary gland type tumors, and papilloma.

Chronic irritation of long duration has generally been regarded as one of the factors in the production of malignant change, and, although it has not been substantiated on a scientific basis, many laryngologists strongly feel that smoking is an important cause for laryngeal metaplasia, particularly in the development of keratosis and carcinoma in situ. The manner in which these chronic irritating factors alter epithelial growth has not yet been uncovered. A history of excessive use of tobacco can be obtained in fully three-fourths of the patients with borderline malignant diseases of the larynx, but when the smoking habits of the general population as a whole are considered there will also be found a large number in which no laryngeal malignancy develops. It would seem that other influences of which we are unaware, connected either directly or indirectly with chronic irritation, are necessary to produce malignant degeneration.

From the Department of Laryngology and Broncho-Esophagology, Jefferson Medical College.

KERATOSIS

The relationship between keratosis and carcinoma has not been definitely established. It is my feeling that keratosis of the larynx is a premalignant lesion in a certain definite percentage of cases, and when the disease persists carcinoma is the usual outcome. In a recent study by Putney and O'Keefe [2] of 125 patients with keratosis of the larynx 57 cases were carcinoma when first seen and 27 cases were first keratosis and later developed into carcinoma.

The clinical term keratosis does not denote a definite and specific entity, for the appearances are extremely variable and changeable. Laryngeal keratosis implies a whitish plaque which may be raised above the surrounding surface or may resemble exudate on the top of the vocal cord. It may appear irregular or smooth and may involve a small portion of the vocal cord or be widespread involving practically the entire larynx. It is not necessarily accompanied by inflammatory change in the surrounding tissue, but when this happens the likelihood of present or future malignant change is increased. An exact histologic diagnosis is essential in differentiating the benign from the malignant keratosis, but the fact that the histologic study reveals a benign lesion is not unalterable proof that the lesion will remain benign.

If it fails to disappear after several weeks when the patient has eliminated all forms of irritation, or if it persists in recurring, the chances are that the lesion will eventually develop malignancy.

CASE 1.—A 66-year-old white man complained of hoarseness of seven months' duration, accompanied by painful swallowing on the right side of the throat and cough. He admitted to smoking two packs of cigarettes daily, a practice which he had followed for many years. On examination, irregular, keratotic areas were observed on both vocal cords, and the histologic report was laryngeal polyp and hyperkeratosis. The lesion improved but did not completely disappear. One year later histologic study revealed infiltrating carcinoma. The patient was treated with irradiation therapy, and has remained well for over four years.

In observing keratosis of the larynx clinically, those lesions which were irregular in their projection above the surface, with a papillary appearance, more often were found to contain carcinoma than those in which the lesion was flat, with no sharp line of demarcation between the keratosis and the normal mucosa.

The management of keratotic lesions of the larynx is not simple. In some cases forceps removal will suffice, but in the majority of cases the management is more difficult. Even though the lesion is benign, when it involves an entire vocal cord or a considerable portion of the vocal cords or extends into the ventricle and onto the ventricular bands, something more than piecemeal removal is needed. Irradiation therapy was given to a group of these patients with beneficial results in that the keratosis disappeared and did not recur. At first rather large doses of x-ray therapy were administered, and in the average case between 5000 and 6000 r was administered as a tissue dose. This amount of irradiation produced considerable postirradiation changes in the larynges, and although the patients were comfortable the laryngeal mucosa never returned to normal. Recently our radiotherapist has reduced the dosage, and on the average approximately 2500 r as a tissue dose was found to be sufficient to eradicate the keratosis, while any permanent irradiation changes in the larynx were avoided.

CARCINOMA IN SITU

Carcinoma in situ is now recognized by pathologists. This alteration in the intraepithelial cellular structure may occur in a normal cord, a cord which is the seat of keratosis, or in an innocuous-appearing pedunculated polypoid lesion. There is considerable discussion about the exact nature of carcinoma in situ, and not all pathologists are in agreement concerning it. This form of malignant degeneration may remain limited to the epithelium for years before there is invasion through the basement membrane. On the other hand it may be the first indication of invasive carcinoma and within a period of a few weeks invasion may occur. A great deal seems to depend upon the sites selected for histologic study, for one may find carcinoma in situ adjacent to invasive carcinoma, or invasion may be present in other portions of the same specimen or in different sections from the lesion. When one receives a diagnosis of carcinoma in situ from the laboratory, he must be sure that the specimen is representative of the entire lesion and that there are no other areas which may contain invasive carcinoma. Once the diagnosis of carcinoma in situ has been established beyond reasonable doubt the problem takes on added complications. Should the lesion be treated as frank carcinoma of the larynx or as a relatively benign lesion? The uncertainties of these preinvasive carcinomas have raised a controversy and no longer can the physician depend upon the pathologic diagnosis of benign or malignant to determine treatment to the exclusion of all other factors. The clinical evaluation of the lesion tempered by the laryngologist's judgment and experience must be relied upon in this respect. I have seen cases in which the noninvasive carcinoma was entirely removed in the biopsy specimen, and as these patients were followed over many years there was no recurrence of the lesion or any subsequent evidence of invasion.

CASE 2.—A 51-year-old white man experienced hoarseness for two months following a cold. He smoked over a pack of cigarettes daily and used his voice excessively. When he was examined by

his physician, irregular, ulcerating lesions with a gray exudate on the surface were observed on the anterior portions of both vocal cords.

He went to one of our large clinics, where direct laryngoscopy with removal of tissue from both vocal cords was performed. The histologic report from each vocal cord was carcinoma in situ. Laryngectomy was advised. Since the patient and his physician desired another opinion, the patient was sent to Jefferson Hospital for evaluation. Under direct laryngoscopic vision the mucous membrane was stripped off each vocal cord so that a portion of submucosa was included in the excised tissue. On histologic examination no evidence of carcinoma was found.

The vocal cords were allowed to heal, and since the patient stopped smoking the larynx has remained free from the disease for four years.

In this type of lesion it is unwise to rush in and perform radical surgery as soon as the pathologist mentions the word carcinoma. When there is histologic evidence of invasive carcinoma, appropriate radical surgical treatment should be instituted promptly, even though one feels that the entire tumor may have been removed by biopsy.

When there is undisputed carcinoma in situ of the larynx, it can be successfully managed without sacrificing the larynx, provided the patient will return for frequent and prolonged follow-up observations. Several methods appear adequate to handle the early, small carcinoma in situ which is limited to a portion of the vocal cord. I prefer simple excision of the lesion down to but not including the perichondrium, preferably through a thyrotomy approach. When the perichondrium is not disturbed, this allows the formation of a better functioning vocal band after the removal of the lesion and in no way jeopardizes the complete eradication of the intraepithelial malignant growth. Irradiation therapy has been reported as curative in a high percentage of these cases. Suspension laryngoscopy with electrosurgical removal of the lesion has also been advocated with gratifying results.

SALIVARY GLAND TYPE TUMORS

Salivary gland type tumors (the so-called mixed tumors) have recently been added to the list of tumors which can be produced in animals by inoculation with a filterable virus. Experimentally, they were multicentric in origin and exhibited malignant characteristics.

Pleomorphism is a prominent feature of this tumor, and any or all varieties of structure may be found in one tumor. They may resemble simple adenomas on the one hand or frankly malignant anaplastic carcinomas on the other. In general the growths outside of the salivary glands tend to be more malignant than those in the salivary glands themselves. Although often classed as benign, they have been demonstrated microscopically to possess the malignant characteristics of nonencapsulation, infiltration of surrounding tissue, recurrence after removal, and occasional metastasis. In aberrant locations it would seem best to consider them all as low-grade malignant adenocarcinomas regardless of the histologic detail. Their slow growth and apparent encapsulation make us inclined to overlook their potentialities. Recurrence has followed excision of the most benign-looking histologic type, while cellular active-looking growths have failed to show malignant tendencies. The usual cause for recurrence is incomplete removal, and in the larynx radical procedures are indicated. Even when the tumor appears clinically encapsulated, microscopic examination commonly reveals extension into the surrounding area, making it unsafe to perform a conservative excision.

In their report of aberrant salivary gland type tumors Putney and McStravog [1] found one case involving the larynx in which recurrence and metastasis to the cervical lymph nodes occurred less than two years after the initial surgery. The patient's chances of a permanent cure would have been much greater if the laryngectomy and block dissection of the cervical lymph nodes which was subsequently done had been performed before recurrence and metastasis developed.

Certain European observers, especially in the Scandinavian countries, have reported favorable results when irradiation and surgery were used together, but in my experience irradiation therapy either alone or in

combination with surgery has failed. Adequate surgical removal has provided a higher cure rate than other forms of treatment.

PAPILLOMA

Interesting observations have been made in animals on the virus-induced papilloma-to-carcinoma sequence. Papilloma virus injected while the animals were contained in the uterus produced papillomatous growths in all the tested animals within two weeks of birth. Carcinoma developed in five of six animals within seven months after the appearance of the papilloma.

In a series of 501 cases of papilloma of the larynx at Jefferson Hospital papillary carcinoma was present in 5%. Of this group there were nine cases in which malignancy developed from a preexisting papilloma, the time interval between the benign and malignant phases varying from 1 to 23 years (Table).

Papilloma Progressing to Carcinoma

Interval in Years	No. Cases
1	1
2	1
4	2
5	1
6	1
9	1
13	1
23	1
Total	9

Activity of the cells in papilloma is frequently observed microscopically even to the point of mitosis. While this change may not alter the maturity of the cell or lead to invasion, this possibility is always present. Even though the origin and clinical behavior of papillomas in children and adults seem to be vastly different, no histologic difference has been noted. The juvenile papilloma was thought to practically never become malignant, until the two cases reported by Walsh and Beamer [3] that underwent malignant change about the age of puberty cast doubt on this assumption.

The papilloma which occurs in adults commonly begins as a premalignant lesion, and is manifested by single or multiple hyperplasia of the epithelium with or without surface keratosis. The papillary growth with well-differentiated epithelium may begin as a low-grade epidermoid carcinoma, and this is particularly likely when keratosis is present on the surface. It is fortunate when these lesions are single and pedunculated so that the entire lesion can be removed and submitted to the laboratory for examination. Our pathologist discovered carcinoma deep within masses of papilloma in three cases. When there are multiple implants and it is impossible to do a biopsy on each separate lesion, carcinoma may exist undetected or develop in other areas not under direct observation. The treatment of simple papilloma alone is unsatisfactory, and when it recurs, removal with continuous histologic search for malignant elements must be practiced. Even when apparent eradication of all papillomatous tissue has been accomplished, the patient should be kept under protracted observation.

In one case malignancy developed 23 years after the initial growth was diagnosed histologically as benign papilloma. During this long span of years the pathologic criteria for malignancy also underwent alteration, with greater emphasis on the individual cell, and this trend influences the modern pathologist in diagnosis more than his predecessor of a generation ago, who was reluctant to make a diagnosis of malignancy unless all of the accepted characteristics were present. Although this change in the field of histologic diagnosis has evolved, there appears to be ample verification of the development of malignant degeneration in previously benign lesions.

CASE 3.—A 34-year-old white man first noticed hoarseness about eight months prior to examination, at which time a pedunculated papilliform growth attached to the anterior third of the left vocal cord was visualized. The entire lesion was removed and submitted to the laboratory, where a histologic diagnosis of papilloma was made. Following this the voice returned to normal and the patient had no further difficulty until 13 years later when huskiness of the voice developed. The voice disturbance gradually progressed almost to a state of aphonia during the following year. At this time, 14 years

after the original papilloma, a large irregular, keratotic papillary mass was removed from an identical location on the anterior third of the left vocal cord and reported histologically as papillary squamous carcinoma. The lesion was removed by laryngofissure, and the patient remained well for the next five years, after which time he was lost to follow-up.

No one form of treatment has received universal acceptance in benign papilloma. In my experience surgical removal, while admittedly leaving much to be desired, has proved more beneficial than other forms of therapy. Roentgen therapy has failed to eradicate the papilloma, and has not prevented recurrence. Surgery has the added advantage of allowing the pathologist to view the recurrent tissue without the complexity of irradiation changes which may confuse the histologic picture and make the discovery of malignant degeneration perplexing. Only rarely will the malignant change be limited to a small area in a single, pedunculated tumor, permitting simple excision of the growth with a wide margin at the base. Commonly when carcinoma becomes evident, it has begun to invade with multiple areas of involvement, so that laryngectomy is necessary.

In benign growths antibiotic and hormonal therapy have been disappointing, and any improvement that resulted seemed to be due to elimination of coexisting infection when the former was used, and regression coincident with adolescence, when the latter was employed, with little permanent effect on the recurrence rate.

SUMMARY

Certain laryngeal lesions, of which the principal ones are keratosis, carcinoma in situ, the salivary gland type tumors, and papilloma, are borderline malignant lesions in that, at times, they are relatively benign and at other times frankly malignant. The development of an invasive malignant phase from a preexisting benign tumor can be substantiated in a definite number of cases. The problem of management is paramount, and it can be simplified to some extent, if any lesion in this category is considered premalignant, and care is taken to ascertain just how benign or malignant each individual abberation is. The determination must be based on both histologic structure and clinical evaluation.

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Lingual Thyroid

Two Cases in Siblings Diagnosed and Treated with Radioactive Iodine

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The purpose of this paper is to review the literature on lingual thyroid and to report the occurrence of lingual thyroid in two brothers in whom radioactive iodine was used for diagnosis and treatment.

HISTORICAL DATA

So far as can be determined the first case of lingual thyroid was described by the English author, Hickman,[1] who in 1869 reported the death by suffocation of a newborn infant. Autopsy revealed a large thyroid tumor at the base of the tongue.

In 1865, Hunt,[2] an American, described a tumor at the base of the tongue in a young woman, and, while he did not recognize this to be a lingual thyroid, the subsequent course of events suggests that he was dealing with such a case. As Hunt described it, the tumor was found in a girl aged 16, situated on the posterior part of the tongue immediately in front of the epiglottis. It filled the fauces completely, but the velum could be readily lifted from it and the finger passed behind its posterior surface; when the tongue was drawn forward and depressed, the parts behind were seen to be entirely normal. In all other respects the patient appeared to be perfectly healthy. Five years before, a physician had examined the throat and at that time the tumor was the size of a small pea on the middle of the posterior surface of the tongue.

The surface of the tumor was very red; large distinct vessels coursed over it; there was no pulsation; it was quite elastic and had an indistinct fluctuating character, not sufficient to indicate the presence of fluid. Opinions as to its nature varied, but favored the view that it was hypertrophy of glandular tissue, probably connected with the part known as the foramen caecum at the apex of the circumvallate papillae. Describing the operation, Hunt says,

> An incision was made into it and a small amount of a peculiar gelatinous-looking substance exuded. But there was no disposition to empty itself. The material was at once examined under the microscope and its character agrees more with the adenoid or glandular variety of morbid growth than any other form.

He did not remove the growth, but strangled it with ligatures and left it to slough away.

The day after operation the patient had some sickness of the stomach, with vomiting later the same day, followed by fever and elevation of the pulse rate to 120. There was great sensitivity to light. The following day the symptoms became more aggravated, and on the third postoperative day she complained of lightness in the head and a sense of smothering and became unconscious. Respirations were limited, the lips were blue and the face was livid; the pulse was small, quick, and irregular. A tracheostomy failed to give relief, and the patient died four hours later. At autopsy no edema of the glottis was found; the larynx and trachea were normal. Hunt then assumed that death was due not to sufffocation but to transference of irritation from the ligated tumor to the respiratory center, and that the vessels of the brain became secondarily involved as indicated by the lightness in the head. Another supposi-

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tion was that her death might have been coincidental.

Comparison of this case with one reported by Haynes [3] in 1912 leads one to believe that Hunt operated upon a lingual thyroid without being aware of its character. Haynes's patient was a woman aged 18, who had a tumor at the base of the tongue, covered with dusky mucous membrane with a network of vessels. It was tense, elastic, not fluctuating, fixed, and not painful. The tumor was removed by the suprahyoid route and was easily shelled out, although it had a dense capsule and the thyroid was vascular and hypertrophic. The postoperative course was interesting. The operation was performed on Nov. 9. On Nov. 24 the patient had an attack of dizziness and fell from a chair apparently in a faint. She complained of pain in the back of the head and said she could not see. During the night she was nauseated and vomited. The following day she felt weak and sick, refused all food and could not leave her bed. On Nov. 26 the temperature was 101 F, pulse 100. She lay huddled in bed in an almost comatose state, although she could be aroused and answered when spoken to. Institution of thyroid therapy brought about gradual and steady improvement in her condition.

It would appear, therefore, that while Hickman [1] is credited with being the first to describe lingual thyroid, Hunt [2] was dealing with one without recognizing its nature.

DEVELOPMENT OF THE TONGUE

The tongue consists of two portions, the body anteriorly and the base posteriorly. The anterior portion is formed from the region between the first and second branchial arch from which projects dorsally the tuberculum impar of His. His felt that the tongue was formed by enlargement of the tuberculum impar; however, later Hammar and Kallius [4] described the tongue as having a paired origin. Behind the tuberculum is a deep evagination of epithelium, the median thyroid evagination, and behind this is a transverse elevation formed by the ventral ends of the second and third branchial arches. The copula, the part corresponding to the foramen caecum, is the residium of the median thyroid evagination. From the copula, the root of the tongue develops.

DEVELOPMENT OF THE THYROID GLAND

The thyroid anlage appears as a prominence of the ventral wall of the pharynx, almost contemporaneously with the formation of the first pharyngeal pouch. It can be recognized in embryos 3 to 4 mm. in length. It soon develops into a vesicle with a constricted hollow stalk, the thyroglossal duct of His. The vesicle becomes a solid mass and enlarges caudalward as a bilobed mass. It becomes isolated from its origin and what becomes later the base of the tongue, by constriction of the upper end of the epithelial cord, which is the obliterated thyroglossal duct. The site of origin after birth is marked as the foramen caecum. The duct usually disappears but may persist as a hollow cord extending from the tongue toward the thyroid body. A cyst containing thyroid tissue may form along the line of this duct. The embryonal thyroid tissue as it descends in the neck enlarges laterally and dorsally. The lateral lobes are united by the isthmus, which represents the main body of the median anlage. In man, the lateral masses undergo reticulation and degeneration and disappear. Accessory thyroid bodies may occur anywhere in the tract along which the median anlage has descended.

However, the thyroid enlarges also by lateral sprouts which may become detached and left behind. Thus the occasional occurrence of isolated thyroid tissue in the region of the upper part of the neck and the neighborhood of the angle of the jaw can be explained.

Aberrant thyroid tissue may therefore be found anywhere along the descent of the thyroid anlage. According to Lahey * it may be at the foramen caecum, in which case it constitutes true lingual goiter; within the tongue, intralingual goiter; below the tongue, sublingual goiter; in front of the larynx,

* References 5 and 6.

prelaryngeal goiter. Two other locations are within the superior mediastinum, and just outside the sternocleidomastoid muscle in the posterior triangle of the neck. Allied accessory thyroid is an extruded adenoma still attached to the thyroid by fibrotic bands. Pseudoaccessory thyroid has an isthmus of true thyroid tissue joining the mass to the thyroid. True accessory thyroid has no connection with the normal gland.

Accessory thyroid is usually understood to be a mass of thyroid tissue existing at a distance from the normally situated gland, while "aberrant thyroid" means that the entire mass of thyroid develops at a site away from the normal location. Lingual thyroid may fall into either category, some cases belonging to the group of accessory thyroids where the gland was also present in its normal location. It is of utmost importance to differentiate between the two when removal is contemplated.

ETIOLOGY

Lingual thyroid is an embryologic malformation; no cause for its appearance is known. Cases have been reported by Van Selms [7] and Willis [8] in which compensatory development of lingual goiter occurred following thyroidectomy and following pathologic destruction of a normal thyroid gland. Enlargement during pregnancy has been reported. In one case reported by Rubeli [9] the enlargement was so alarming that Caesarean section was necessary; following the termination of pregnancy the growth subsided. Because disturbance of the thyroid is more apparent in the female in puberty and during pregnancy, it may be assumed that as many cases occur in males which escape attention because the gland is less active in the sexual sphere. Undoubtedly the cases reported form a relatively small percentage of those that exist.

INCIDENCE

The most comprehensive review of the subject of lingual thyroid is that made by Montgomery,[10] who tabulated all known cases reported up to 1936. He studied reports on 231 possible cases, but actually only 144 are accepted by him as authentic. Of these, 142 were cases in which the lesion occurred on the dorsum of the tongue; in 2, thyroid tissue developed in the body of the tongue. Montgomery's criteria of authenticity are as follows:

1. Examination of the removed specimen should reveal thyroid gland tissue.
2. In lieu of this requirement, thyroid insufficiency should supervene after removal of the nodule.
3. The lesion should appear in the substance of the tongue between the epiglottis and the circumvallate papillae.

Buckman [11] in 1936 included 246 cases in his tabulation, including one of his own. The discrepancy in the numbers reported is apparently due to the different criteria used, inasmuch as Buckman included at least 32 cases in his series which did not meet the criteria of Montgomery.

A search of the literature since then reveals approximately 17 additional cases, including 2 reported by Timmons [12] in 1951 and 1 reported by Waters [13] in 1953.

The condition occurs with much greater frequency in women. In Montgomery's series of 144 cases, 118 were females, 17 were males, and sex was not recorded in 9. The age at onset of symptoms was between birth and 61 years in females, between 12 and 54 years in males. It is interesting to note that in only three cases of the female group and one of the male group were symptoms present at birth or during infancy, because in the two cases herein reported the symptoms were noted during the first month of life.

SYMPTOMS

Symptoms vary from slight voice change and moderate interference with deglutition or slight hemorrhage, to extreme dysphagia, choking spells, orthopnea, severe dysphonia, and exsanguinating hemorrhage. The symptoms most often observed are dysphagia, dysphonia, and dyspnea, and they appear to be due to either obstruction of the pharyngeal fossa, pressure upon the epiglottis and orifice of the larynx and esophagus, or to

increase of the distance between the epiglottis and the circumvallate papillae with impairment of muscle function; or to a combination of the three.

Montgomery [10] in his survey of 144 cases found the symptoms in the order of frequency to be

Dysphagia—slight in 17, moderate in 24, and severe in 31.

Dysphonia—slight in 28, moderate in 29, and severe in 6.

Dyspnea—slight in 13, moderate in 16, and severe in 11.

Hemorrhage—slight in 5, moderate in 11, and severe in 8.

Pain—slight in 2, moderate in 6, severe in 2; however, 3 of the patients who complained of pain had carcinoma.

Fullness or tightness in the throat—slight in 2, moderate in 3.

Dysphagia was the most frequent symptom and caused the most distressing disturbance, undoubtedly because the lesions were a greater barrier to the passage of food than movement of air, and because they interfered more with the mechanism of deglutition than with either respiration or phonation. Dysphagia was so great in a few patients that severe inanition resulted.

The voice changes ranged from a slight nasal quality to marked muffling and inability to articulate clearly.

Dyspnea, in most cases, was not a prominent symptom even with large lesions. In the severe cases it was usually troublesome when the patient was in the prone position. Three infants died apparently of suffocation.

Hemorrhage from the mouth, originating presumably from the lingual node, was observed in 24 cases, 4 of whom were males; 1 was believed to have a carcinoma of the lingual thyroid nodule. The amount varied from slight coughing or spitting of blood to the tremendous discharge of 2000 cc. In three female cases, bleeding from the mouth was in relation to the menses.

Fullness or tightness of the throat was present in 5, "catarrh" or sore throat in 5, snoring in 5, fits of coughing in 1, a sense of swelling in the neck in 7, and pain in 10.

APPEARANCE

The typical lingual thyroid is a rounded hemispherical mass which occupies the median line of the base of the tongue between the foramen caecum and the epiglottis. It is covered with normal intact mucosa with a varying degree of vascularity and is reddish in color. It is circumscribed and distinct from the glossal structure on a broad base. It reaches the epiglottis but is not adherent to it; the epiglottis may be pressured backward. It may vary in size from a tumor which fills the pharynx to a small island within the base of the tongue. It is semisoft or firm, elastic, not fluctuating, painless, not tender, and does not blanch on pressure.

PATHOLOGY

Lingual thyroid is a benign tumor, but may, because of grave hemorrhage, alarming dyspnea, or constant dysphagia, present a serious threat to life. Malignant degeneration is rare. Montgomery [10] found in the 144 cases reviewed, 4 with proved malignant change, three of which metastasized to the cervical glands and lungs. All four cases occurred in males past the age of 35. No indubitable cases of lingual thyroid carcinoma have been reported in women.

In Montgomery's series there appears to be no definite relationship between the presence of a lingual thyroid and the basal metabolic rate. Four patients showed signs of hyperthyroidism, while signs of thyroid insufficiency were present in 21 cases. However, a number of autopsy reports on cretins who died early in life of athyreosis, the only thyroid tissue found was that at the base of the tongue (Aschoff,[14] McCallum,[15] Erdheim [16]). Adult cretins have been described by deBoncourt,[17] Brunner,[18] Didier,[19] Harvey,[20] Krassnig,[21] Lindt,[22] Leulier,[23] Matti [24] and Rebattu.[25] In no instance was a thyroid gland present in the normal location, and the only thyroid tissue found was at the base of the tongue.

Postoperative myxedema has followed extirpation of a lingual thyroid in many cases.

DIFFERENTIAL DIAGNOSIS

Lingual thyroid is to be differentiated from other benign tumors which may appear at the same site.

Angioma is purplish in color, soft and compressible, blanching under pressure and refilling on release.

Amyloid is so rare that it is of no significant interest; only one case has been reported and may have represented an advanced stage of degeneration of a thyroid nodule.

Adenoma is a firm tumor, embedded, which resembles the thyroid tumor. Severe cases of lingual thyroid have been classified as adenomata.

Cysts of the thyroglossal duct may occur in this location. The fluid contained in the cyst imparts a fluctuant sensation as compared to the solid quality of the thyroid tumor. Aspiration of the contents shows clear viscid material and may produce collapse of the wall.

Dermoid cysts and tumors are classified by some writers with thyroid tumors of the tongue, since they have a common origin. Differential diagnosis may not be possible before removal.

Chondroma and osteoma are exceedingly rare.

Fibroma is fairly common; it is a smooth hard mass, often pedunculated, without the varicosities of the thyroid tumor.

Gummata are usually multiple, often ulcerated, with associated adenopathy. Serologic and therapeutic tests will confirm the diagnosis.

Hypertrophy of the lingual tonsils is flat, verrucose, superficially located, and distributed anteriorly to the foramen caecum. It is not circumscribed as is the thyroid tumor, and is of softer consistency. It is not vascularized.

Lipoma is soft and yellowish, lacking the firm and elastic quality of the thyroid tumor and usually occupying the anterior portion of the tongue. The lesion is rounded and circumscribed.

Lymphangioma of the tongue is soft and compressible, pale, and may be translucent.

Papillomas may be sessile, but are more often pedunculated. They are reddish to pale, soft, and tend to favor locations other than the base of the tongue.

Of the malignant tumors, epithelioma and lymphosarcoma are to be differentiated. Carcinoma of the tongue usually involves the anterior half or the tip along the margins. It may appear at the base, but usually to one side at the base of the tonsil. It is indurated and infiltrating.

DIAGNOSIS

Diagnosis could be made from the typical appearance of lingual thyroid. The time-honored method of diagnosis by biopsy and microscopic examination has, in general, produced complications out of proportion to the value received in the case of lingual thyroid nodules. Goetsch [26] advises definitely against biopsy since his experience with one case in which infection and sloughing and prolonged inflammatory reaction supervened. These factors made subsequent surgical resection difficult and unsatisfactory.

The availability of radioactive iodine during the past 10 years has opened an entirely new approach to the study of thyroid problems in general and to the identification of aberrant collections of thyroid tissue in particular. The application of this radioisotope to thyroid investigation depends upon two factors; first, the truly remarkable biologically unique property of thyroid tissue to trap and concentrate iodine, and, second, the fact that radioactive iodine, which behaves just like stable iodine in the body, emits a powerful gamma radiation which can be measured outside of the body.

On the basis of the foregoing knowledge of an ideal tracer situation, Feitelberg [27] described a method for the demonstration of radioactive iodine accumulation in lingual thyroid tissue, using a Geiger counter. Timmons [12] has also used the technique successfully in two cases and considers it diagnostically superior to the notoriously difficult biopsy procedure.

TREATMENT

Certainly not all lingual thyroid tumors call for active treatment. Many of the tumors appearing at the time of puberty must represent physiologic hypertrophy and might better be handled as are analogous hypertrophies in the normally situated gland. Many may represent the only functioning thyroid tissue present. While conservative treatment consisting of rest, and symptomatic treatment with the administration of thyroid and extract, may be preferable to hasty surgical intervention, surgery may be imperative in cases of marked dyspnea, dysphagia, dysphonia, and recurrent or grave hemorrhage. If surgery is necessary, the presence or absence of the normally situated gland should be determined. While older authors advise an exploratory dissection of the thyroid region of the neck for this purpose, the use of radioactive iodine will now answer the question as to whether thyroid tissue is present in its normal location, and whether one is dealing with an aberrant thyroid or merely accessory thyroid tissue at the base of the tongue.

If activity can be demonstrated at the location of the normal thyroid gland, one may then consider removal of the lingual goiter if the severity of the symptoms necessitates intervention. If, on the other hand, no activity can be demonstrated at the normal site, complete removal of the lingual nodule is contraindicated. If the symptoms are severe, partial resection can be done and any hypothyroidism occurring subsequently must be treated with the administration of thyroid by mouth. In the two cases described in this report, the use of radioactive iodine resulted in substantial decrease in size of the lingual goiter with complete relief of symptoms.

If surgical removal is required, this can be done by either the oral route or through the hyoid or transbuccal route. Electrocoagulation has also been used successfully for shrinkage.

Tracheostomy may or may not be required, depending on the individual case.

REPORT OF CASES

CASE 1.—G. A. C., a male infant, was first seen at the age of 4 months on Sept. 8, 1950. The chief complaint was dysphagia and noisy breathing, which had been noted by the mother shortly after birth. The infant had noisy respiration and choking spells, more marked during feeding, at which time there was difficulty in swallowing. He had gained weight because he was given smaller feedings at shorter intervals. He was referred by the family doctor who suspected a laryngeal obstruction.

The patient was a normal full-term child, had measles at the age of one month and was given immune globulin; there was no other significant history. One older sister was living and well.

Examination revealed a well-developed, 4-month-old white boy, not acutely ill. He seemed to have some difficulty in breathing; however, no suprasternal retractions were noted. His voice seemed clear. Physical examination was essentially negative except for findings in the throat. The tonsils were small. A mass was seen in the midline, which at first glance appeared to be behind the tongue. It was seen only momentarily when the child gagged and then disappeared downward. This was followed by a choking spell lasting several seconds, following which unobstructed, though somewhat noisy, respiration was resumed. The first impression was that of tumor of the epiglottis.

The child was admitted to Presbyterian Hospital on Sept. 13, 1950. Laboratory and x-ray findings were within normal limits. On Sept. 14, laryngoscopy was performed by Dr. S. A. Friedberg and me, general anesthesia with nitrous oxide being used. A mass the size of a walnut was found at the base of the tongue in the midline. It was firm, elastic, and was covered by a grayish, firm capsule, which was moderately vascularized. It seemed to push down on the epiglottis; however, the larynx could be exposed without difficulty and appeared to be normal. An attempt was made to secure a specimen for biopsy with a punch forceps; this proved impossible because no bite could be obtained on the firm capsule. Aspiration of contents of the mass was attempted unsuccessfully. After repeated attempts a small superficial piece of tissue could be grasped with the forceps and this was sent to the laboratory.

A tentative diagnosis of lingual thyroid was made. The microscopic report read: "The material consisted of a section through buccal mucosa. This is covered by a layer of stratified squamous epithelium which is relatively normal in appearance. In the underlying tissue there are found a few thin-walled vascular channels and in one of the biopsies there is a long strip of elastic cartilage, which is well defined and normal in appearance. In the deeper lying strata there is no evidence of tumor in this

material. Diagnosis: Normal appearing buccal mucosa from the region of the base of the tongue."

It was evident from this report that only the mucosal covering of the capsule was obtained. It was then decided to give radioactive iodine to confirm the diagnosis, and also with the hope that it might shrink the mass and obviate the need for major surgery.

On Sept. 15, at 6:30 p. m., 100μc of I^{131} in 30 cc. of milk was given, and on Sept. 16 the Geiger counter demonstrated concentration in the area of the normal thyroid. With this region shielded, activity about the angle of the jaw and floor of the mouth was also increased. Efforts to place the counter directly on the lesion were unsuccessful. From these findings it was felt that a diagnosis of accessory thyroid tissue at the base of the tongue was justified (Dr. R. Gordon Brown).

The patient was discharged from the hospital, and, when seen in the office two weeks later, the mother reported improvement in the infant's condition. He was free from choking spells and seemed to breathe better and take nourishment better. When he returned to the office on Feb. 27, 1951, he was completely asymptomatic. Inspection and palpation of the tumor revealed that it had shrunk in size. No further treatment was deemed necessary.

When again examined on Feb. 5, 1954, the patient was a well-developed 4-year-old boy without any symptoms of dyspnea or dysphagia. Inspection of the throat showed no evidence of a tumor mass. Indirect laryngoscopy revealed an area about 5 mm. in diameter at the base of the tongue, which appeared to be different from the surrounding lymphoid tissue. It was barely elevated, had a smooth surface and was slightly indurated to palpation. This was apparently the residuum of the lingual goiter.

Case 2.—T. L. C., brother of the first patient, was seen on April 14, 1952, at the age of 2 months. There was again a history of choking spells, more marked during feeding, which began shortly after birth. Because of the similarity of symptoms to those manifested by the brother two years previously, the mother immediately suspected a similar condition and brought the child to the office.

This patient was also a normal full-term child. Examination revealed a well-developed 2-month-old white boy in no acute distress. The findings in the throat were identical to those seen in his brother. A round mass, seen at the base of the tongue, was firm, elastic to palpation, and again examination was followed by a choking spell.

The patient was admitted to Presbyterian Hospital on April 16, 1952. Laboratory and x-ray findings were within normal limits. On April 17, a direct laryngoscopy with use of vinyl ether (Vinethene) and ether anesthesia was performed by Dr. S. A. Friedberg and me. The operative report is as follows: "The child followed the same pattern as his brother who was examined two years ago because of respiratory obstruction and dysphagia. There was a similar finding of a small round mass in the midline at the base of the tongue. This mass was extremely firm and its capsule could not be penetrated by ordinary biting instruments."

Biopsy report (Dr. G. M. Haas): "Five slides show serial section of a biopsy at the base of the tongue. They show stratified squamous epithelium with a small amount of subepithelial connective tissue containing lymphocytes, small amount of normal salivary type glandular tissue and a few blood vessels. There is no evidence of tumor in this material. Diagnosis: Negative buccal mucosa." Again a tentative diagnosis of lingual thyroid was made and, in view of the great improvement which followed the use of radioactive iodine in the patient's brother, it was decided to use it in this case also.

On April 17, at 5 p. m., 100μc of I^{131} was given in 30 cc. of milk. On April 18, studies with a Geiger counter revealed a normal concentration in the area of the thyroid. With this region shielded there was an increase in activity about the angle of the jaw and floor of the mouth. Diagnosis of lingual thyroid of the accessory type was made (Dr. R. Gordon Brown).

The patient was discharged from the hospital and was not seen again until Jan. 30, 1953, at which time the mother reported that his choking spells began to lessen about four weeks after the application of I^{131} and gradually disappeared. Examination at this time showed the lingual nodule still present, but apparently of smaller size. The child was asymptomatic and had no difficulty with breathing or swallowing. On Feb. 2, 1954, inspection of the throat failed to reveal any abnormality. Palpation revealed a small indurated area at the base of the tongue.

The child is now a healthy-appearing 2-year-old boy.

SUMMARY

A review of the literature on lingual thyroid is presented.

Two cases of lingual thyroid occurring in siblings, believed to be the first such cases, are presented.

The use of radioactive iodine for diagnostic and therapeutic purposes is reported.

Arcade Building.

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Threshold Finding in Pure Frequency Audiometry

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The literature on hearing testing contains relatively few specific directions regarding the threshold finding procedure in pure frequency audiometry. Some "procedures" often quoted will be found on examination to contain little specific procedural information. It is the purpose of this paper to provide specific steps for obtaining the auditory threshold.

All individuals working in the area of hearing testing would probably testify to the basic importance of a general philosophical or theoretical orientation regarding the nature of hearing and the nature of testing. In spite of the importance of such an orientation, the person who is actually conducting audiometric examinations as an important part of his diagnostic procedure will find a general philosophy an incomplete guide when he is faced with a specific client. In addition to the orientation, a step-by-step procedure is of vital concern. Such a procedure must be useful for the audiometrician in the daily conduct of the hearing examination.

In this report the author proposes to set forth a step-by-step procedure which will be operational with a large number (certainly not all) of persons. There is a lack of experimental evidence to substantiate many of the procedures here proposed. Nevertheless, in the face of a real clinical problem where some action must be taken, specific directions must be set forth to solve the problem at hand. When the specific experimental evidence is available to warrant changes in these suggested procedures, the author will be the first and happiest to adopt the experimental findings.

From University of Illinois.

PROBLEM

The problem in clinical audiometry is to find the hearing threshold—to accomplish this accurately and rapidly—with as little tiring of the client and frustration for the tester as possible. The following suggestions are made with the view to accomplishing this important clinical goal.

METHOD

The author has previously outlined [1] a step-by-step procedure for part of the audiometric examination. This technique is labeled the "standard" technique. This label is in contrast to other techniques, and not because there is any real measure of agreement or "standardization" of the steps specifically involved:

1. The subject is seated (facing 45° or 135°) away from the audiometer and tester so that he receives as few visible cues as possible during the testing.

2. The subject is asked to respond when he hears the stimulus tone by raising his hand. He is instructed to hold up the hand as long as he hears the tone; he is to put the hand down when he no longer hears the tone.

3. Six frequencies are tested in the following order: 1024, 512, 256, 1024, 2048, 4096, 8192 cycles per second.

4. The ear to be tested first is predetermined randomly unless the subject hears better in one ear than in the other; in the latter case the better ear is tested first.

5. Threshold finding procedure.

(*a*) The initial stimulus is presented at a hearing loss of 30 db (as marked on the audiometer dial).

(*b*) If the stimulus is heard, the level is decreased in 10 db steps until it is inaudible.

(*c*) From inaudibility, the level is increased in 5 db steps until the stimulus is audible.

(*d*) From audibility, the level is decreased in 5 db steps to inaudibility.

(*e*) The tone of lowest intensity to which the subject gives three consecutive responses is considered the threshold, and recorded.

(*f*) If the initial stimulus at 30 db is not heard, the level is increased in 10 db steps until there is a response. From this point, the same procedure of down in 5 db steps and then up in 5 db steps is used to determine the thresholds.

6. After the threshold for each frequency has been determined, the other ear is tested following precisely the same procedure.

COMMENT

This set of steps, 1 to 6, gives a procedure to be followed in arriving at the audiometric threshold. However, there are several further questions (procedural refinements), which are not answered by the steps set forth. The following suggestions are made in an effort to add further information for efficiency in achieving the hearing threshold.

1. The tester should check the audiometer before seeing each client. Check the audiometer against the known hearing acuity of the tester.

2. Choice of ear to begin with is made as follows:

If the client is known to have a hearing loss, start with the better ear.

Ask client in which ear he hears better. If ear is stated, start with that ear.

If no information as to better ear is available, and person indicates no preference, then choose ear randomly.

3. With known hard of hearing individuals, or when testing in a school for the deaf, start the stimulus at a level of 60 db rather than 30 db.

4. Know and observe carefully the output limits of the audiometers. Mark the limits presented during the test; use a special symbol when stimulus is presented at audiometric limits.

5. Time variation of stimulus presentation. Hirsh [2] says there is no difference in giving the individual an additional benefit from a cue to the stimulus. However, the stimulus presented at regular temporal intervals gives rise to distinct and specific "anticipatory responses" in many individuals. This is a problem separate from the matter of cues "helpful" to the client. These anticipatory responses are familiar to every experienced audiometrician. To the naive tester these responses can give very faulty thresholds. One way to avoid such anticipatory responses is to vary the temporal interval at which the stimuli are originated.

6. Ask the client to use his finger to touch the earphone in which the stimulus is heard. It is my opinion that such a signal is a most reliable form of response.

7. Be certain that the *finger* signal indicates clearly the ear in which the hearing is localized. Touch the finger to the earphone in which the stimulus is heard. Some lateralization problems may be avoided by making certain that the individual indicates the ear in which he hears the stimulus.

8. Be certain also that the finger response corresponds to the preservation of the stimulus. In this way the tester has two indications of the reception of the signal—when the signal begins and when it ends. It can be most distressing to the tester to have the person persist in holding the finger up long after the stimulus has been removed.

9. Subliminal cues should be watched for and should be used whenever possible; that is, accept the subliminal cue as if it were an overt response. Follow through the whole procedure and evaluate this response as if it were overt. Every practiced audiometrician is familiar with the type of partial finger extension, clenching of the fist, furrowing of the brow. I recommend that in most such instances the response be accepted.

10. The client should be instructed that whenever in doubt about the presence of the stimulus, he should respond as if he heard it. The tester will evaluate such a response with the other information he has available about the presence of the stimulus.

11. The client should be kept alert.

12. The client should not receive a stimulus longer than one-half second to one second. Keep the stimulus short.

13. Special procedures may have to be adopted with particular groups, especially brain-injured children. Also, small children may require special attention.

SUMMARY

The problem of giving an audiometric test to a person who presents no special testing problems is disarmingly simple. However, the beginning tester is apt to gain not only confidence but a false sense of his ability to give audiometric tests for all sorts of problems and to all sorts of individuals. The suggestions which are presented here are not intended to cover some of the more complex aspects of the hearing-testing situation. I have not mentioned bone conduction; the practices covered here are intended primarily

for air conduction although some of them are equally applicable in the bone-conduction situation. Bone conduction brings special considerations of masking and lateralization, with which I shall not deal here.

It is fervently hoped that these suggestions will be accepted in the spirit in which they are given. These are specific suggestions to deal with specific problems in the hearing testing situation. I recognize that few of these procedural steps are based on experimental evidence. No one wishes any more strongly than I do that such experimental evidence were available. Until such information is forthcoming, the suggestions herein contained are offered as interim guidance for the practical clinical situation. I will be the first to welcome evidence to indicate that there are better ways to accomplish the end set forth as the problem of this article.

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Reconstruction of Soft Tissue Side of Nose

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Reconstruction of an absent ala and lateral wall of the nose can be accomplished in many ways, the way usually being dictated by the degree of lost substance and the condition of the local surrounding tissues.

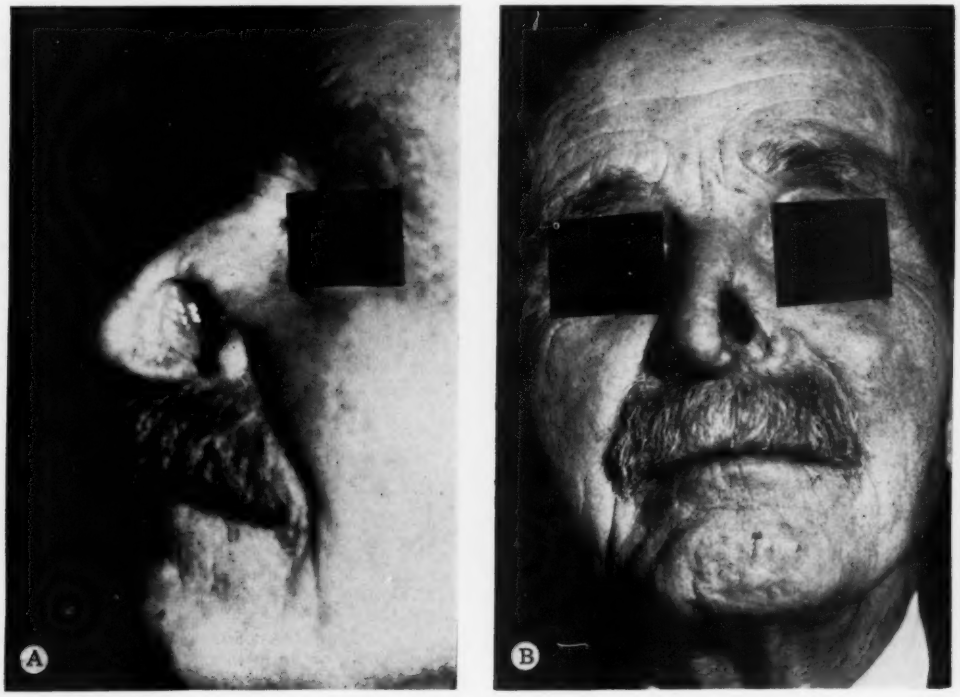
If the margin of the ala be missing and it be not too large, it may be replaced by a composite free graft taken from the ear. This replaces the surface skin, supplies a pleasing margin and the lining, all in one procedure. It is an excellent, easy way of correcting such a small defect. Should the marginal defect be large, such a free graft will not take. The area being too large to fill by this method, a sliding, or pedicle, flap must be used.

When a larger area is involved, say the entire soft tissue lateral wall, including both the lateral cartilage and the lateral wing of the ala, one then has three problems: 1. The defect must be covered by skin. 2. This covering must be lined within the nose to prevent scarring and secondary contraction. 3. The reconstructed side of the nose usually requires some firm support, such as cartilage, to prevent it from sagging into the nose.

These large defects may be replaced by borrowing skin from the forehead, arm, cheek, etc. Most of these methods require several stages to attain their goal and are useful in selected cases.

This past year, an 84-year-old man consulted me regarding plastic repair of the soft tissues of the nose. Three years previously,

Fig. 1.—*A*, Lateral view showing preoperative defect. *B*, Front view showing preoperative defect.



he had been operated on for basal-cell carcinoma involving the soft tissue of one side of his nose. There was no evidence of recurrence, and he was most anxious to have the area closed by something other than a patch. The patient was a poor surgical risk, and a method of filling in the defect using a one-stage procedure was sought.

Dr. F. deQuervain [1] closed a small hole in the lateral wall using a septal flap hinged along the dorsum. This method supplied the lining and cartilage support, which was covered by a local skin flap. The principle was ideal, as all our requirements were fulfilled in one procedure.

Searching the literature further, I found that Dr. Varaztad Kazanjian [2] had presented the reconstruction of an ala also, using a septal flap. After the septal flap had healed in position, he replaced the outer mucous membrane with a skin graft. Another article [3] by him showed a reconstruction of the entire soft tissue side of a nose utilizing a septal flap and a median forehead flap. This was most practical but required two stages, the second to cut the pedicle and return the stalk to its bed.

This patient, being elderly, had relaxed facial tissue, so it seemed advisable to use a septal flap for a foundation and lining which could be covered by a cheek flap taken adjacent to the nasolabial fold.

Employing local infiltrative anesthesia which contains epinephrine, 1 : 50,000, the operation may be performed with a minimal of bleeding. I prefer this over general anesthesia for most operations about the face because of the hemostatic effect of the epinephrine.

The margin of the defect is first freshened and prepared to receive the septal flap. All tissue is saved that is vascular; the margin which is dense scar may have to be sacrificed.

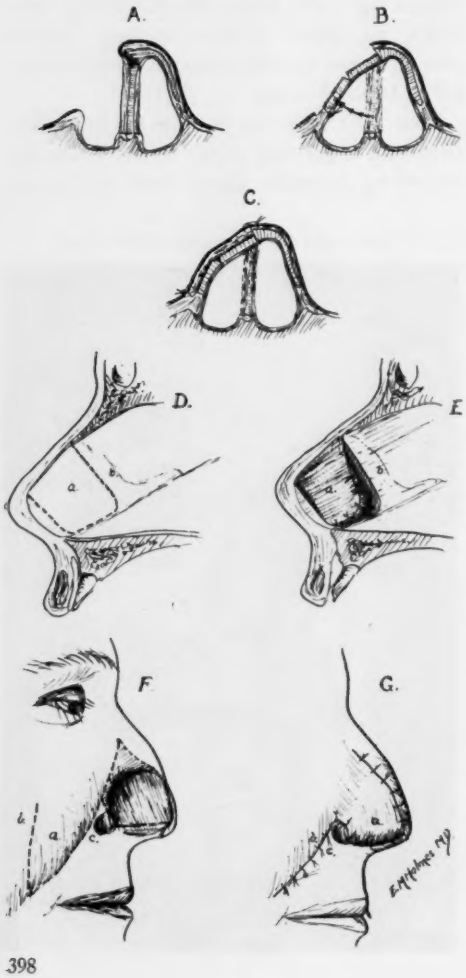


Fig. 2.—*A*, Cross section of the nose showing defective ala and "V" trough of cartilage to be removed parallel to dorsum, which will break spring and permit hinge of mucous membrane.

B, Full section of the septum, which has been hinged at the top, permitting the cartilage and mucosa on the inside to swing laterally, where it is attached to the under surface of the defect. The mucosa on the outer surface has been removed in anticipation of a recipient graft. Note that a wedge has been removed from the cartilage just at the tip to permit the mass to swing laterally, and an incision has been made through the cartilage down to the membrane halfway down to permit the cartilage to buckle outward.

C, The septal lining and cartilaginous support has been covered with a full-thickness skin graft, as shown in Figure 2*F* and *G*.

D, The dotted line indicates the through-and-through incisions of the septum. Note that a thin margin of cartilage is left in contact with the anterior maxillary spine and comes up to the tip, thus holding the tip in position.

E, The through-and-through septal flap has been sutured to the lateral wall, the remaining defect being left alone.

F, The defect and the incisions made to create a cheek flap as well as the lower margin of the septal flap.

G, The completed operation. The tongue of the flap has been cut to fit the inside of the nostril and has been sutured to the free border of the septal flap.

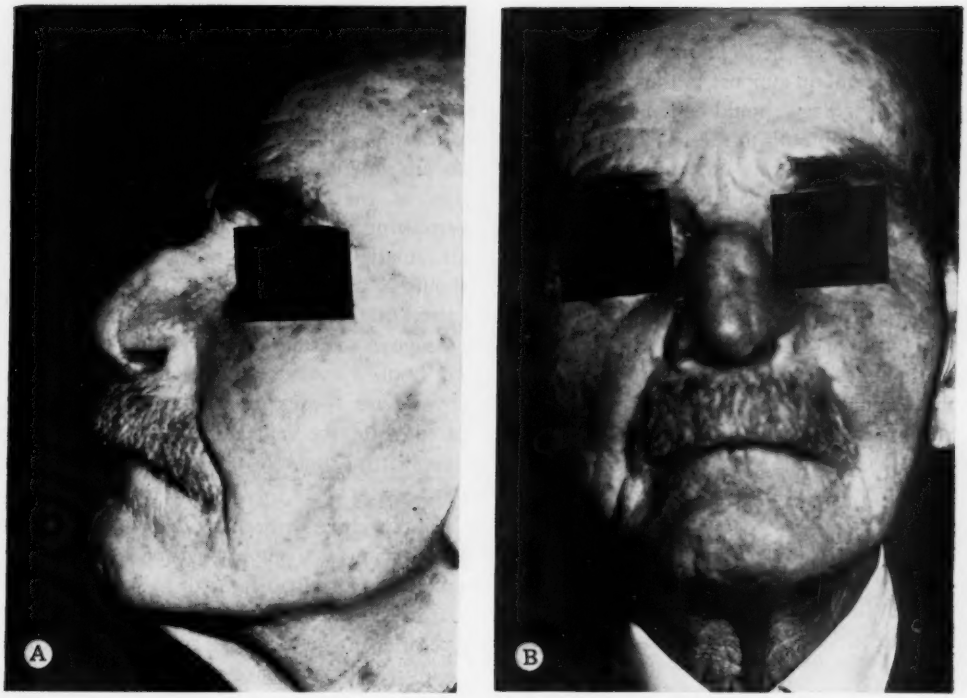


Fig. 3.—*A*, Lateral view showing postoperative results. *B*, Front view showing postoperative results.

Fig. 4.—*A*, Preoperative front view of second case. *B*, Postoperative front view of second case.

The septal flap is quickly obtained, but one criterion should be kept in mind, as it will help to maintain support to the tip. A narrow strut of cartilage should be left from the premaxillary spine up to the nasal tip (Fig. 2*D* and *E*). Saving this not only keeps the tip up but prevents the columnella from shortening or being pulled up into the nose. An incision is made through-and-through the entire septum from the dorsum down to the vomer, then posteriorly along the vomer ridge to the tubercle, then up again to the tip of the nasal bones. This permits it to hinge along the dorsum. Where the lateral cartilage fuses with the septum, it creates a spring, which tends to pull the septal flap back into position and to create a concavity to the flap. To overcome this, a wedge of cartilage should be removed parallel to the dorsum, creating a "V" trough sufficiently wide to permit the septum to hinge out into position (Fig. 2*A*). The blood supply is maintained through the mucous membrane on the intact side, which acts as the hinge. The mucous membrane on the exposed, outer surface is next removed, as the denuded cartilage will be covered with a skin flap. In performing this step, I leave the perichondrium in contact with the cartilage, removing the surface membrane only. The septal flap is now sutured into the defect using 0000-absorbable surgical (gut) sutures, with the knots in the nasal cavity (Fig. 2*E*).

If the side of the septal flap is too flat, it may be made to have a convex contour by cutting a line parallel to the dorsum through the cartilage only, and then a packing inside the nose will hold it in position until it heals in this position (Fig. 2*B* and *C*).

A skin flap is now planned out adjacent to the nasal defect, employing skin of the cheek so that the resultant scar will fall along the nasolabial fold (Fig. 2*F*). It must be wide enough to extend from the dorsum of the nose to the base of the defect and long enough to fold upon itself to form the margin of the ala and line itself up to the septal graft. The skin of the cheek is widely undermined, permitting easy closure employing 4-0 or 5-0 atraumatic sutures (Fig. 2*G*). The nose should be packed with petrolatum (Vaseline) gauze around a rubber tube to permit the passage of some air and then a pressure dressing applied outside. No attempt is made to cover over the exposed bone or remaining cartilage of the septal defect, as this covers over with a mucosa in a couple of weeks in most cases.

This simple procedure has fulfilled our three criteria of supplying the nasal defect with lining mucosa, cartilage support, and normal-appearing skin covering, all in one operation. It may be necessary at a later time to adjust the ala marginal skin for improvement in symmetry, but such is a minor procedure.

The result is gratifying not only cosmetically but functionally, and in selected cases reduces a multistage procedure to a single one (Figs. 3 and 4).

330 Dartmouth St. (18).

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Gonadotropism

An Etiologic Factor in Migraine and Migraine Variants

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INCIDENCE

In a study of migraine and migraine variants conducted by Grimes and reported by Cecil,[1] out of 15,000 persons examined, 1200, or 8%, were affected. No age, social, intellectual, or economic group is immune.

Migraine or migraine variants occur more frequently in the female, about 70% of the total.

Morton and co-workers [2] found that 62% of the crimes perpetrated by women were committed in the premenstrual week.

REASONS FOR THIS INVESTIGATION

Considerable clinical data available strongly suggest that gonadotropism must be considered an etiologic factor in such common conditions as migraine, histaminic cephalgia, tension, and other pain mechanisms due to vasodilation of the cerebral arteries or other vascular dysfunctions.

The exact gonadopathy is not clear. I believe, however, that there is frequently both a gonadal imbalance and a deficiency. I further believe that there is more frequently an androgen, rather than estrogen, deficiency in both sexes.

In the female most imbalances probably exist because of hyperfolliculinemia, rather than hypogonadism or hypergonadism. The latter would tend to produce a balance between androgen and estrogen, even though there was an excessive amount of both in the blood.

From The Department of Otolaryngology, University of Kansas School of Medicine and The Research Clinic.

I was encouraged to carry on this clinical investigation into the causes and treatment of vascular headaches because of certain physiological conditions—all pointing to gonadotropism—having at least a direct influence in these painful head and neck syndromes. They are as follows:

1. The premenstrual headache—migrainous in type—has long been recognized as a clinical entity.

2. The relief at times of migraine and other types of headaches during pregnancy suggests that the body is supplied with some substance, or at least a better balance of the existing substances, that did not exist in the nonpregnant state.

3. Menopause frequently ushers in an era of joyous relief for some and poignant distress for others.

FUNDAMENTAL PRINCIPLES OF SEX STEROID IMBALANCES AND DEFICIENCIES

The adult man produces a small amount of estrogenic substances, which largely comes from the adrenal cortex, perhaps a small amount even from the testicle. The adult woman produces significant amounts of androgen in the adrenal cortex. In the treatment of many conditions in both men and women several authors have suggested a ratio of 20:1 of testosterone and estrone. This ratio has not as yet been finally determined. There is great individual variation which must be carefully considered, requiring flexibility of ratios lacking in available pharmaceutical preparations. It is, therefore, incorrect to speak of separate male and female hormones. No normal woman is without masculine secretion. No man is without female secretion. Someone has remarked, "no Adam without an Eve." Each possesses the indifferent gonad at an early stage of

germinal epithelium, which has the same structure in both sexes.

Many investigators have postulated that the androgens probably have a direct effect on the pituitary gland by stimulation, which may in turn increase ovarian activity in the same manner. This would seem to rationalize the extensive use of the androgens in the female patient. The theory of antimetabolitic activity, of neutralization between estrone and testosterone, due to chemical structural resemblance, has been espoused by Woolley,[3] but this theory has not met with favor among many endocrinologists.

Ryan [4] believes that the symptoms of headaches are so closely related to those physiologic processes which are peculiar to the female as to require special consideration. There is, during the early portion of the menstrual cycle, a gradual increase in the level of estrogen circulating in the blood, an increase which persists until just before the onset of menstruation. This fact makes deplorable the use of estrogenic substances to control the pain of the premenstrual migraine and other vascular headaches. This treatment often intensifies the distress of the patient by further increasing the imbalances already existing between estrogen and androgen. It also augments the nervous hyperirritability—tension—which in turn produces a change in the size of the cranial vessels and a change in the muscular tonicity of the skeletal muscles of the head and neck.

TREATMENT OF MIGRAINE AND MIGRAINE VARIANTS *

The judicious use of the combined sex steroids, with the concomitant correction of other imbalances and deficiencies, has been found by me to offer the best hope for the prevention of recurring attacks of various types of migraine and migraine variants. The administration of a single sex steroid has been found worthless, except in the gonadectomized patient, and, even then, the addition of a small amount of the opposite sex steroid is advantageous. The enhanced therapeutic value is probably due to an additive or synergistic action, perhaps both. There is probably a harmonious interaction between the sex steroids, other hormones, vitamins, and minerals. An encapsulated preparation of this type is given once or more times daily, when indicated, to each patient, male or female, suffering with migraine or any of the migraine variants.

The balance between androgen and estrogen probably varies from 25:1 to 10:1.

The androgenic hormones are chiefly of value as powerful protein anabolic hormones, for their retention of nitrogen and for their ability to build up proteins. These are, therefore, the most universally accepted steroids for use in both sexes.

A long-acting androgen is given intramuscularly in 50 mg. doses, or less, once or twice a month.

The concomitant administration of the vitamin B complex and ascorbic acid with all hormones is obviously advantageous for their catalytic and synergistic action, one with the other.

An adequate nutritional regimen is essential.

CONCLUSIONS

Migraine or migraine variants in some form were present in about 8% of 15,000 patients examined by Grimes and reported by Cecil.[1] About 70% of all vascular headaches occur among women.

Morton [2] found that 62% of crimes perpetrated by women were committed in the premenstrual week. Relief of vascular headaches during pregnancy and after the menopause strongly suggests that gonadotropism is probably a dominating factor in the etiology of these headaches.

* Comment: Specific therapy in migraine would, of course, be highly desirable, but it is practically unfeasible, owing to plurality of etiology. Gonadotropism is only one etiologic factor. Autonomic imbalance, hypothalmic dysfunction which disturbs vascular rhythm, imbalance in the acid-base mechanisms, etc., must all be evaluated before specific therapy is considered. One must, therefore, recognize the fact that the cause of any pathologic state is the sum total of all conditions required to produce it. To put reliance in only one specific therapeutic procedure is but to invite imperfect and, at times, disheartening results.

Clinical studies by me seem to confirm the opinion of the physiologists and endocrinologists, that there is no separate male or female hormone. The male produces a small amount of female hormone, while the female produces a significant amount of the male hormone, mostly in the adrenal cortex.

These same clinical studies show a variable ratio between androgen and estrogen from 25:1 to 10:1. Clinical judgment and therapeutic trial must determine the individual needs for each patient.

It has been observed that the younger, more virile males with migraine headaches have at times required a slight increase in estrogen in the encapsulated preparation to obtain a gonadal balance. The effeminate type requires an increase in androgen to accomplish the same objective. I believe that gonadal imbalance, often made worse by the injudicious use of single sex steroids, has not only in the past been responsible for many gonadal therapeutic failures but also has been the cause of many miserable and, at times, serious sequelae.

The concomitant administration of vitamins and minerals by mouth in an encapsulated form and the correction of other imbalances and deficiencies, as advocated by Revici and Welt,† has been found by me to be highly desirable.

The androgenic hormones are given intramuscularly for their powerful protein anabolic effect in both the male and the female.

This report, founded on a small series of cases, is not statistical. In the beginning the results were mostly unanticipated, because the combined hormones were being administered as a part of therapy to correct multiple general deficiencies.

I wish to emphasize that the criteria used in evaluating the results were entirely subjective. However, nearly all patients reported a noteworthy improvement in their painful episodes. The attacks of some continued, but the intensity, duration, and frequency were lessened. The headaches were "bearable."

Others reported that the attack would start but did not quite "come through." Still others, the most gratifying of all, stated they now had only an "Aspirin headache." Nearly all related an improvement in general well-being, less nervous tension, and less fatigue.

No virilism was noted in this study. I believe that this was due to the gonadal balance.

Libido was infrequently reported.

Patients must be observed not less often than once a month.

In younger persons all gonadotherapy should be frequently interrupted as a means of determining the necessity for its further use. Gonadotherapy is seldom used in juvenile migraine. In older persons, however, its continued use may be indicated as a supplement to replace the hormones not synthesized, owing to a declining physiologic activity of the gonads. The good nutritional regimen must not be discontinued, because as long as the endocrine glands are supplied by an adequate and congenial chemical medium they will synthesize their own hormones and pharmaceutical supplementation becomes unnecessary.

It must be stressed that much more study, continued observations, and clinical evaluations are essential before any final or unequivocal statements can be made anent the etiology and treatment of migraine.

Incomplete investigation, however, and inadequate therapy can only terminate in therapeutic failure.

Headaches of psychogenic origin which simulate migraine must be carefully ruled out.

I am still experiencing some defeats in the treatment of migraine. I am confident, however, that recent scientific work means real help for today's migraine patient. The number of complete failures is sharply reduced; the successes are perceptibly increased.

Dr. William H. Masters, of Washington University, allowed me to review his comprehensive writings on the subject of gonadal dysfunctions.

† References 5-8.

Revici, Stoopen, Ravich, and Welt ‡ gave assistance in the understanding and in the management of migraine and other painful episodes. They effectively stress the connection of an acid-base imbalance to painful syndromes. This phase of the subject could not appropriately be discussed in this report. It is, however, suggested that the reader refer to their contributions and other equally thought-stimulating reports published by them and their associates at the Institute of Applied Biology, Brooklyn.

The oral preparations used in this study are a multivitamin, mineral, and hormone preparation containing ethinyl estradiol and methyl testosterone (Gevrine), supplied by Lederle Laboratories Division, American Cynamid Company, and a multivitamin preparation with estrogens and d-desoxyephedrine hydrochloride (Mediatric) supplied by Ayerst Laboratories, Inc. The long-acting androgen Depo-Testadiol, containing 50 mg. testosterone cyclopentylpropionate, 2 mg. estradiol cyclopentylpropionate, for parenteral use, and 5 mg. chlorbutanol in each cubic centimeter, was supplied by The Upjohn Company.

1110 Professional Building.

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Experimental Defects in the Ear and the Upper Airways Induced by Radiation

GEORGE KELEMEN, M.D., Boston

The large amount of knowledge which has accumulated to form a basis for developmental pathology as "a new and thrilling discipline of biology and medicine" (Gruenwald, 1949) has provided evidence that ionizing radiation affects developing and rapidly growing tissues. Recognizing the fact that different tissues react in different ways, the very first observers, half a century ago, tried to tabulate the organs and their constituents according to their degree of susceptibility to injury by x-rays.

Irradiation of pregnant animals, at various stages of development of the embryo and fetus, has shown that the time of injury was the chief factor in determining the pattern of the malformation, although the dose influenced the severity of the defect.

A series of papers (see references in Hicks, 1953 and 1954) disclose the mechanism by which irradiation brings about malformations and the developmental factors that determine the critical periods for these patterns. In the rat, no deformities followed irradiation in the first eight days of gestation. Embryos irradiated in the earliest somite stages (ninth day) developed severe head defects involving the forebrain, olfactory apparatus, eyes, skull, and face, including nose and, to some extent, the jaws and the base of the skull. To this pattern the term anencephaly is applied; others called the condition pseudencephaly. Embryos irradiated in the 10th-day period (about three to eight somites) developed malformations generally limited to the eyes ("anophthalmia") and sometimes involving the pituitary. As described in detail (Hicks, 1953), these embryos sustained severe acute radiation damages, but largely recovered from them. In particular, their faces, upper respiratory passages, and temporal bones developed virtually normally despite this damage. Embryos irradiated in the 11th-day period (about 10 to 20 somite stages) showed developmental changes (Fig. *A*) more subtle than the grotesque changes of the 9th day, and those near term developed definite hemorrhages in the head region. The latter could not be accounted for as artifacts produced by the manner of killing the fetus, namely, drowning (asphyxiating) it in fixative or sectioning it with a razor blade. It is this group of animals whose developmental characteristics near term are given in the following account; within this framework the present paper reports on changes found in the hearing organ and in the upper airways, with discussion of the possible relationships of these experimental results to mechanisms of human malformations.

Growth and maturation of organ systems of the developing rat, as in any embryo, do not present a uniform progression. The hearing organ of the rat completes only about two-thirds of its developmental course in utero. The gestation period being 21 days, another 10 to 11 days are needed for full maturation of the organ of Corti. In a painstakingly detailed study starting from the first day of postnatal life, Wada (1923) followed the growth of Corti's organ and associated structures in the rat. He found

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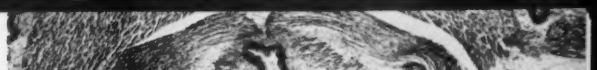
From the Department of Otolaryngology, Harvard Medical School, and the Department of Otolaryngology, The Massachusetts Eye and Ear Infirmary.



that between the 9th and the 12th after-birth day the tunnel of Corti appears and the rat can hear. Mature relations are established at about the 20th day. Two waves of change run within the membranous cochlea: one from the axis to the periphery and the other from the base to the apex. The rat hears usually on the 12th day, three days before the eyes open. The largest cells in the spiral ganglion reach their maximum size at 20 days: The rat hears, therefore, before these cells reach their full size.

Unfortunately, no comparable, exact "time-table" study exists concerning the details of the development of the hearing organ in the rat before birth.

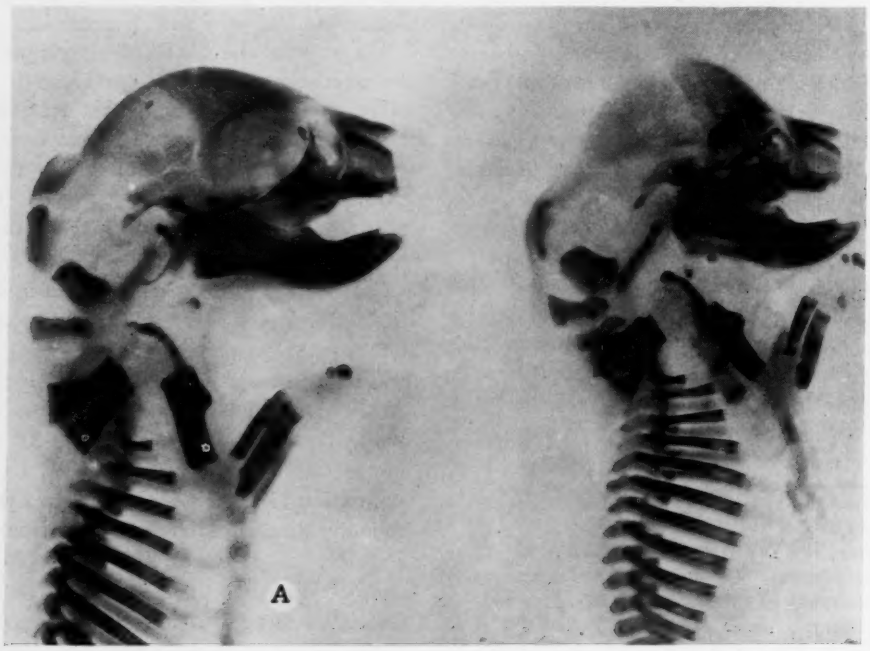
MATERIALS AND METHODS

The malformed animals, the data for Table 1, and Figure *A* were most generously made available by Dr. Samuel P. Hicks, from the Department of Pathology, Harvard Medical School, and New England Deaconess Hospital, Boston. For this particular study, animals which had been irradiated on the 9th to the 13th gestational day were chosen, and the final observations made at term. The findings, in terms of development, range from the stage of the beginning of the 1st somite to about the 40th. They represent, for the most part, the large number of animals used by Dr. Hicks in previous studies in which the nervous system was the principal interest, as well as current material from animals whose younger fetuses were more precisely aged at the time of irradiation by the procedure of sampling members of the same litter at successive intervals during development, starting a few hours after irradiation.

The 21 rats studied in detail near term with serial sections came from an inbred strain of the Harvard University Biological Laboratories, originated from Wistar Institute stock; or from an inbred Sloniker strain.

Irradiation was carried out with General Electric Maximar therapy units. The factors were 200 and 250 kv., 10 ma., 3-mm. inherent Al filter, target distance of 50 cm., about 50 r per minute. The whole pregnant animal was exposed. The apparent dose delivered to the animal, as measured in air, and the useful working range varied from 150 to 300 r, usually 150 to 200 r. Near term, the mother was killed by thoracic spinal section; the members of the litter were removed and fixed in Bouin's fluid or 10% formalin (see Hicks, 1953). Serial sections of the head, and intermittent or serial sections of the body, were prepared after paraffin embedding; they were stained mostly with hematoxylin and eosin.

Fig. *A*.—Malformation of the skull resulting from irradiation on the 11th day of gestation in a newborn rat (right); normal rat is on the left. (Supplied by Hicks.)



The technical preparation was carried out by Mrs. Elizabeth C. Newcomb and Mrs. Regina C. O'Brien, and the photomicrography by Mr. Donald C. Whitee.

OBSERVATIONS

A certain number of the 21 animals showed no remarkable changes in ear, nose, or larynx (10th-day animals, as noted above). Others showed alterations restricted to one side, but generally alterations were present bilaterally. Without attempting a statistical evaluation, the outstanding features of the results of irradiation on the 11th day are given here in a comprehensive picture.

The larynx presented in all cases an entirely normal configuration (Figs. 1 and 2). Cartilages, muscles, and mucous-membrane lining showed undisturbed development. The only unusual finding, seen in a single animal, was represented by edematous folds protruding from the anterior aspect of the thyroid cartilage above the insertion of the vocal cords (Fig. 3). The walls of the ventricle of Morgagni were still without glandular apparatus.

The nasal cavity (Fig. 5) contained some scattered secretion, already showing signs of resorption associated with leucocytic infiltration. Of more significance were endocranial bleedings in the region of the cribriform plate impinging on the region of passage of the fila of the olfactory nerve (Fig. 4). The turbinal surfaces were encountered in different phases of swelling by engorgement: Their regular play seems to start immediately with the entering of air. Engorgement was

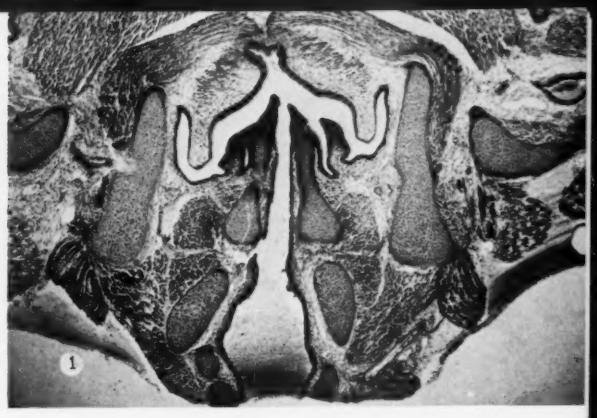


Fig. 1.—Larynx, general view, frontal section. Cartilages developed, musculature well outlined. Morgagni's ventricle reaches the level of the upper edge of the thyroid cartilage; no glands in the ventricular wall. Reduced from × 40.

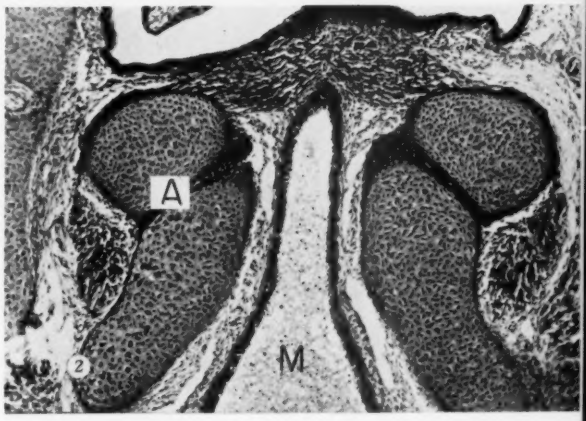


Fig. 2.—Larynx, frontal section. Cricoarytenoid articulation (*A*), well formed, surrounding well-differentiated musculature. Glottis closed by mesenchymal tissue (*M*). Reduced from × 100.

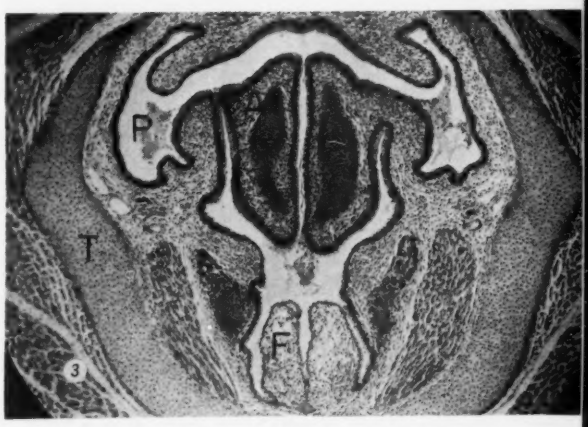


Fig. 3.—Larynx, near-horizontal section. Blood in the pyriform sinus (*P*). Arytenoids (*A*), closing the intercartilaginous section of the glottis. Two edematous folds (*F*) project from the inner aspect of the thyroid cartilage (*T*). Reduced from × 40.

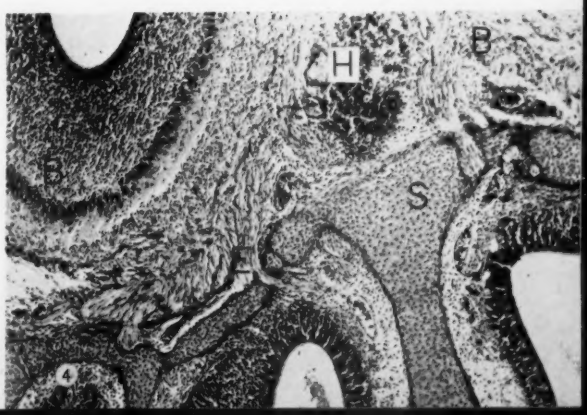
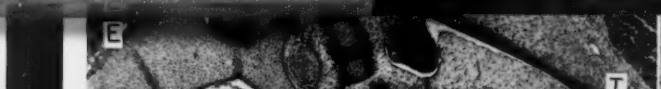
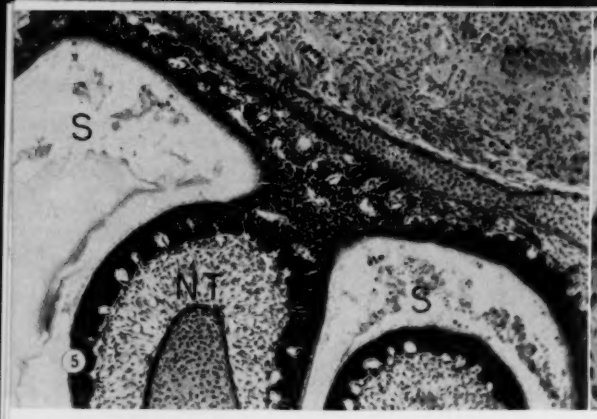
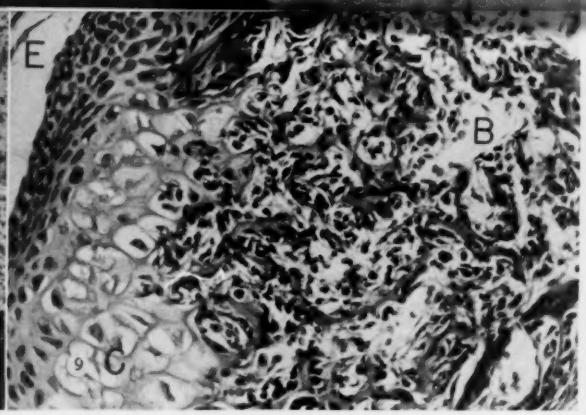
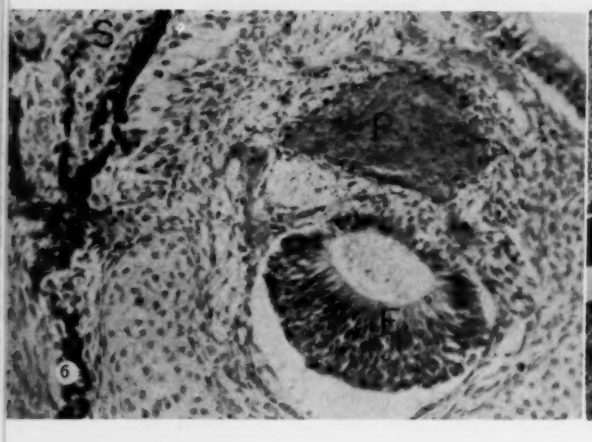


Fig. 4.—Olfactory organ. Hemorrhage (*H*) between the two olfactory bulbs (*B,B*), just above the nasal septum (*S*). Right and left of the septum, fila (medial group) of the olfactory nerve, piercing the cribriform plate of the ethmoid (*E*). Reduced from × 80.









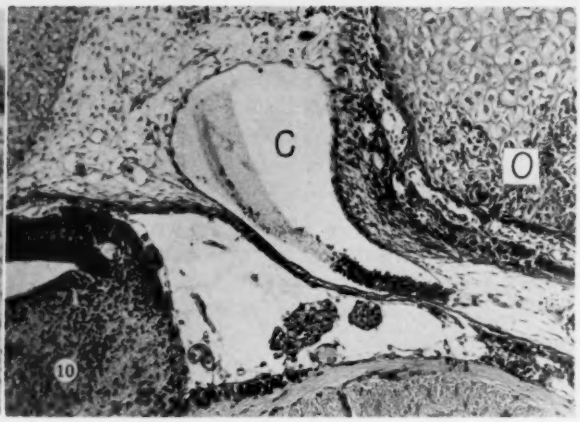


Fig. 5.—Nasal cavity. Secretion (*S*) in resorption at both sides of the nasoturbinal (*NT*). Reduced from × 150.

Fig. 6.—Vomeronasal organ (Jacobson) in cross section, at the base of the nasal septum (*S*), with engorgement in its vascular plexus (*P*); *E*, sensory epithelium; reduced from × 250.

Fig. 7.—Eustachian tube, wide open; its advancing epithelium initiates the pneumatization of this area of the tympanic cavity forcing back by pressure the mesenchymal tissue (*M*); reduced from × 250.

Fig. 8.—Eustachian tube; pharyngeal orifice with engorgement in the venous plexus (*P*) of its posterior lip; reduced from × 250.

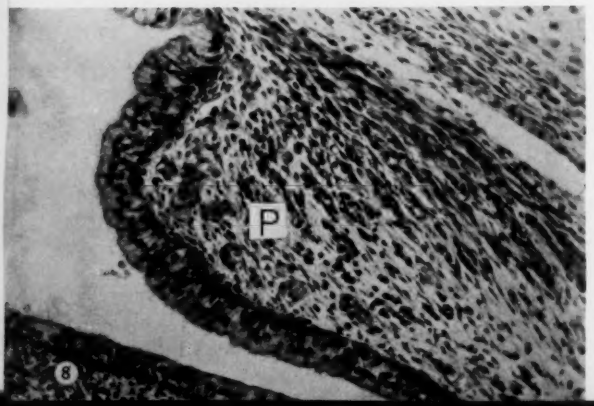
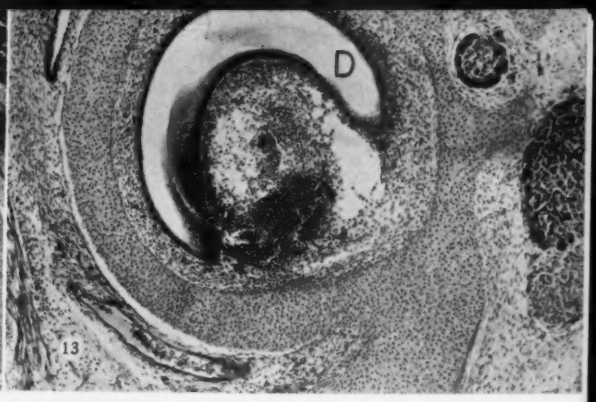
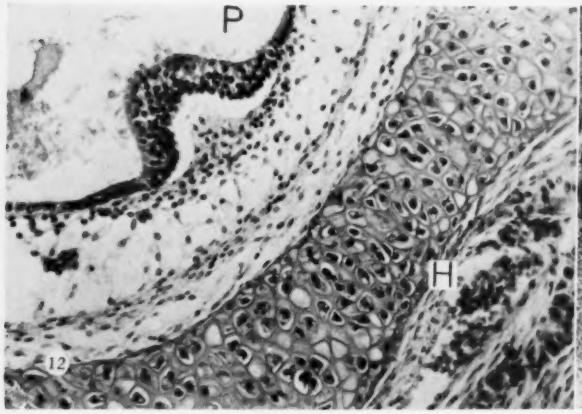


Fig. 9.—Ossification center in the presphenoid bone; disturbance of the regular formation of lamellae by suffusion with blood. *E*, epithelial lining of the epipharynx; *C*, cartilage; *B*, new bone. Reduced from × 250.

Fig. 10.—Arachnoidal, intradural, and subdural hemorrhage, with formation of a cyst (*C*) outside the dural opening of the inner acoustic meatus. Hemorrhage in an ossification center (*O*) of the labyrinthine capsule around the mouth of the meatus. Reduced 45% from mag. × 150.





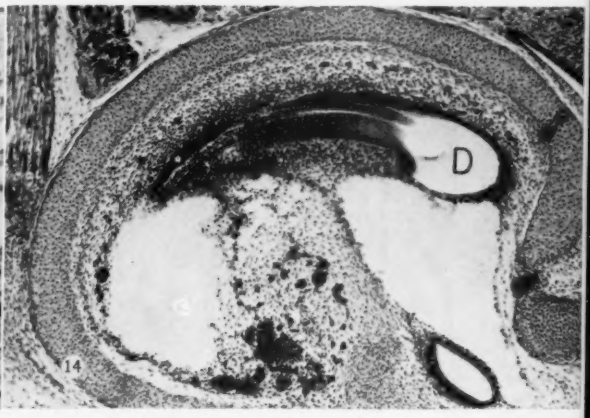


Fig. 11.—General view of the inner and the middle ear, in parahorizontal section. Internal acoustic meatus (*M*), with intraneural hemorrhage in the eighth nerve. Cochlea (*C*) in paramodiolar section. Sacculus (*S*) with macula sacculi; utriculus (*U*) with macula utriculi and crista IV; endolymphatic sac (*E*). Above the stapes (*ST*) the middle ear. In the perilymphatic space beginning resorption of the mesenchyma; endolymphatic space already free. Middle ear still filled by mesenchyma, with pneumatization initiated around the ostium of the Eustachian tube (*T*). Reduced 45% from mag. × 40.

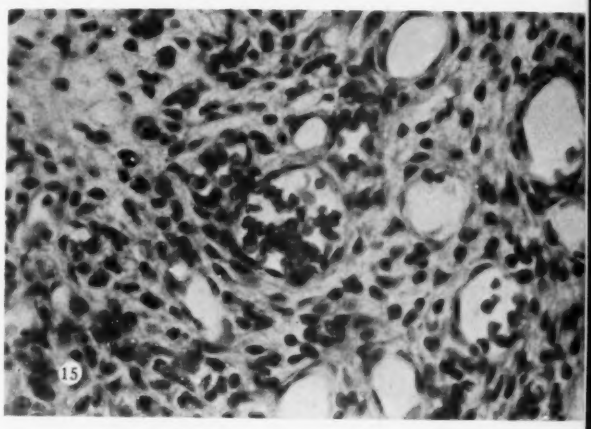
Fig. 12.—Internal acoustic meatus, with perineural hemorrhage (*H*) around the eighth nerve. *P*, posterior crista; cupula torn away (artifact). Reduced 45% from mag. × 250.

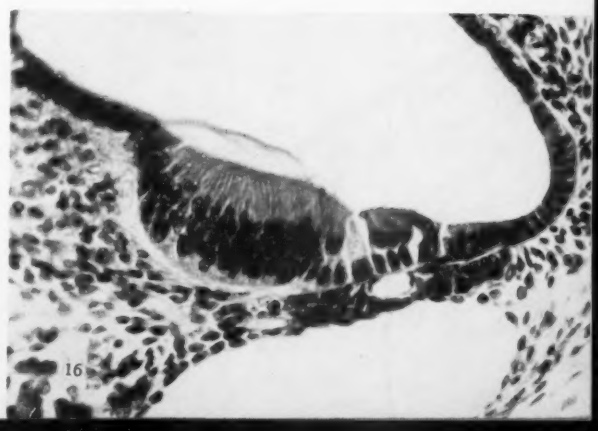
Fig. 13.—Tangential section of the cochlea. Massive hemorrhage in the (connective tissue) modiolus. *D*, cochlear duct. Reduced 45% from mag. × 80.

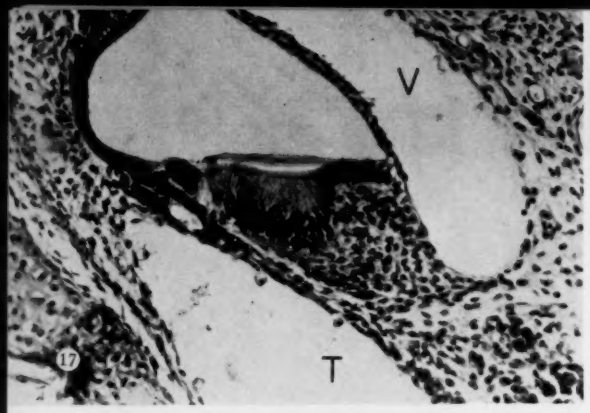
Fig. 14.—Tangential section of the cochlea. Scattered hemorrhage in the (connective tissue) modiolus. *D*, cochlear duct. Reduced 45% from mag. × 80.

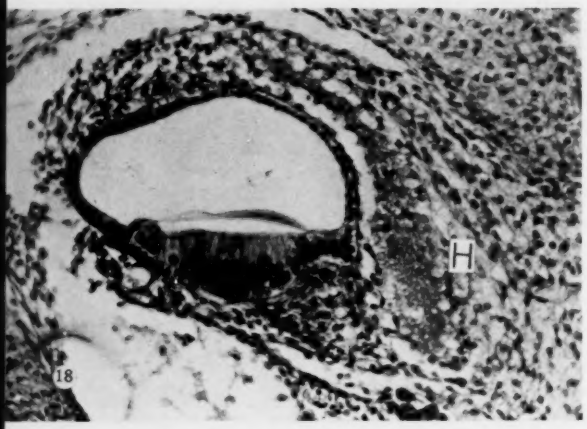
Fig. 15.—Engorgement in the mesenchymal vessels of the (connective tissue) modiolus; reduced 45% from mag. × 500.

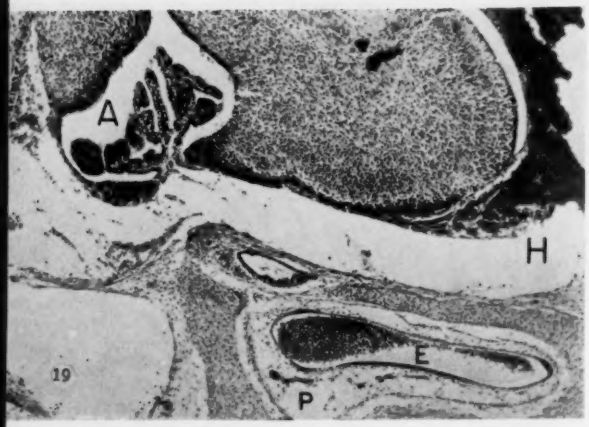
Fig. 16.—Corti's organ of an anencephalus, undisturbed as to its developmental stage. At the left side the greater (medial), at the right side the lesser (lateral) epithelial ridge; the former covered by the tectorial membrane. Reduced 45% from mag. × 500.

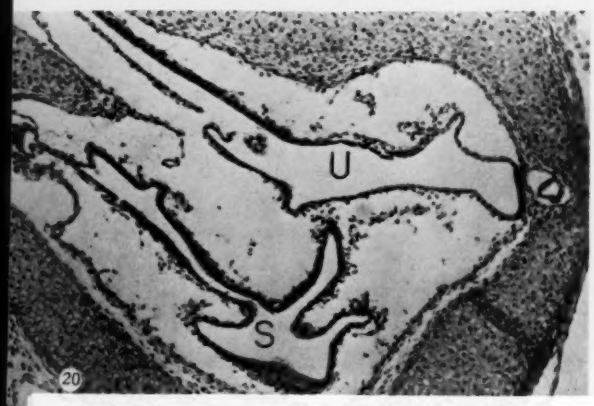












the rule in the vascular plexus of the organ of Jacobson (Fig. 6) and in a similar location, namely, the vascular plexus in the nasolacrimal duct.

In the basisphenoid the ossification center showed severe suffusion with blood (Fig. 9).

The position of the petrous portion of the temporal bone was changed; in shape it had become flattened and depressed. The pyramids were lying nearer to a horizontal plane, and the angle between the two petrous bones was widened.

The auditory tube was always patent in its entire length (Figs. 7 and 8). The lumen contained some granular debris and occasional leucocytes. In its pharyngeal mouth, in the prominent posterior lip, a highly engorged conglomerate of vessels was outstanding (Fig. 8).

The middle ear (Fig. 11) was completely filled by mesenchyma, except in a small pneumatized area around the tubal corner. Small extravasates and engorgement were seen in the mesenchymal tissue. The undamaged cartilaginous ossicles were on their way to regular definite differentiation.

The region around the dural orifice of the internal acoustic meatus showed many large subarachnoidal hemorrhages, which penetrated into the meatus. Sometimes serum-filled cysts were seen here (Fig. 10). The lateral sinus was enormously enlarged by engorgement.

The inner ear was subject to manifold disturbances. The inner acoustic meatus carried the only ossification center of the entire otic capsule near its dural opening; here the newly formed bone was suffused

Fig. 17.—Cochlear duct of the second coil, with tympanic (*T*) and vestibular (*V*) scalae partly formed by mesenchymal resorption (normal control); reduced 45% from mag. × 250.

Fig. 18.—Section comparable to Figure 17. Hemorrhage (*H*) filling the partly formed vestibular scala. Reduced 45% from mag. × 250.

Fig. 19.—Arachnoidal hemorrhage (*H*). Endolymphatic hemorrhage (*E*) and perilymphatic engorgement (*P*) in the posterior semicircular canal; *A*, arachnoid villi.

Fig. 20.—Compression of the endolymphatic tube by the edematous perilymphatic space; the former already entirely, the latter only partly free of mesenchyma. *S*, sacculus, with part of the macula; *U*, utriculus. Reduced 45% from mag. × 150.

with blood (Fig. 10). The subdural hemorrhage, penetrating the meatus, caused compression of the eighth nerve when it had occurred between the meatal wall and the nerve (Fig. 12). In other cases the localization of the extravasate was intraneural, disrupting portions of the nerve stem (Fig. 11).

Within the cochlea (which shows two and one-fourth turns in the rat) hemorrhage was generally found in the connective-tissue modiolus, in massive (Fig. 13) or scattered (Fig. 14) form. Engorgement of the vessels, including the vas spirale, was observed (Fig. 15). The scalae were already partially formed after resorption of the mesenchyma (Fig. 17); and the free portion of these perilymphatic spaces was filled with blood (Fig. 18). The development of the organ of Corti was remarkably unaffected (Figs. 16 to 18). Slight disorganization of the basilar membrane was seen a few times, but is disregarded here as being possibly artificially produced. The tectorial membrane floated freely over the papilla (Figs. 16 to 18) or turned back around its central attachment, as on a hinge. The cochlear duct was without exception free of hemorrhage. The membrane of Reissner held its normal position; a slight bulging (Fig. 18) was produced in certain cases, probably by the pull of the receding mesenchymal tissue overlying it.

The relatively unchanged condition of the end-organs was equally conspicuous in the vestibulum. Here the extravasates were rarely located in the endolymphatic space (Fig. 10), and then with preference in the posterior canal; they were found more frequently in the perilymphatic space. A finding of consequence was perilymphatic edema with compression of the endolymphatic tube (Fig. 20). This was observed around the sacculus and the utriculus as well as in the region of the semicircular canals. The extravasate was located only in the endolymph, or only in the perilymph, or in both spaces. The perilymphatic area was crossed by engorged vessels.

COMMENT

The combination of irradiation at midterm with observation at term was chosen to provide a comprehensive picture of the possible changes. The period under review should later permit more detailed elaboration. The 9th- to 10th-day period of normal gestation brings rapid changes in the differential development of the organs. That this initial series of observations should end at term was indicated by the high incidence of infections in ear and upper airways that start immediately after birth in "normal" animals. In man, the middle ear of the newborn infant is seen to contain, a few hours after birth, masses of pus cells and bacteria; induced infection can go on to labyrinthitis and meningitis (literature in Hemsath, 1936). Aspiration material is always present in cases of pneumonia of the newborn infant (Kaldor, 1933). Pathological findings in the nasal cavity of young rats occur in about 40% of so-called "healthy" controls (Kelemen and Sargent, 1946). In young albino rats, Nelson and Gowen (1930, 1931) observed, by macroscopical dissection, an incidence of 30% middle-ear infection, while this figure increased rapidly with advancing age. Relative protection may be offered by the mesenchyma, which in the rat practically fills the middle ear at birth. However, pathological conditions caused by inflammation occur here in the first days of extrauterine life, a period when, in man, they are extraordinarily frequent (Alexander, 1926).

To correlate the findings in the ear with those in the other organs a timetable of ir-

TABLE 1.—*Developmental Defects Resulting from Irradiation of Rat Embryos in Utero* *

Gestational Day of Irradiation	Defect
9	"Anencephaly": severe facial, forebrain, and skull defects
10	"Anophthalmia": slight pituitary anomalies; virtually normal skull, temporal bone, nose
11	Beginning encephalocele; hydrocephalus; microphthalmia; defects of skull, lower spine, upper spinal cord
12	Porencephaly and microcephaly; encephalocele; spinal cord, retinal, and spine defects
13 to term	Microcephaly (various patterns); toward term, cerebellar anomalies

* Data were obtained from Hicks, S. P.: Am. J. Roentgenol. **69**: 272 (Feb.) 1953.

radiation is given (Table 1), summarizing the events found during the course of the entire series as it is known to the present time. Table 1 emphasizes the severe head deformities, which dominate the picture in the immediate surroundings of the ear. Correlation of the data of this table with the ear study shows at once a point of maximum relevance: the integrity of the end-organs in the cochlea and vestibulum, in the immediate vicinity of the most severe cranial abnormality.

The lower cranial nerves of the anencephalic animal are well developed, and so is the eighth nerve complex, because the primordia of these parts are not yet in a radiosensitive stage on the ninth day. Damage is sustained on the 10th and 11th days to the auditory placode and primitive neuroblasts that give rise to the middle and lower cranial nerve complexes, but there is virtually complete recovery. Malformation of the base of the skull in the 11th-day period is therefore attributable primarily to alterations in the differentiating mesenchyma that is vulnerable at that period.

The nervous apparatus appeared to be normal peripherally from the acousticofacial complex. According to Donaldson (1924), in the rat fetus of 35 mm. (about at birth) the ampullae of the semicircular canals are well innervated, with good differentiation of the sensory and the supporting cells. In the waltzing guinea pig, Lurie (1939) saw nerve fibers still going to the basilar membrane, with no organ of Corti present. That this organ developed normally in the vicinity of a disorganized central nervous system shows a high-grade developmental independence from the nervous system. The organ of Corti, the maculae, and cristae exhibited their appropriate developmental stage.

A striking example is offered by the findings in a case in which the irradiation resulted in a very high degree of deformity, anencephaly. One ear showed no abnormality; the other showed hyperemia in the connective-tissue modiolus, perilymphatic edema around the sacculus and utriculus; there were edematous folds in the larynx. The other parts of the body were found to be normal.

In one instance, a remarkably resistant organ of Corti was found amid the destructions caused by a spontaneous serous labyrinthitis in the guinea pig (Kelemen, 1930). Novotny (1951) applied x-rays to the inner ear of adult guinea pigs; here, again, microscopic structure and function remained intact. Block (1952) exemplified with a clinical case the damage which can be inflicted to the temporal bone by x-rays, but this represented adult conditions. Potter and Adair mention oxycephaly: microcephalus with a tower-shaped head, as found fairly frequently among the offspring of women exposed to roentgen therapy in the course of their pregnancy.

Hertwig (1944) has made a study of waltzing in mice induced as a genetic characteristic by x-rays. Increased lymph pressure resulted in cyst formation, starting from the cochlear duct and reaching dimension to form an obstacle for the development of cerebrum and cerebellum. In the present experiments acquired developmental cysts were similarly seen, especially around the dural orifice of the internal acoustic meatus, but their size was not conspicuous. In the quinine experiments of Dederding (1953) on guinea pigs, phenomena of endolymphatic compression were frequent.

It might be mentioned that Girden (1935) produced in dogs a transient gain in hearing acuity with roentgen rays. In no case was this function impaired, although skin burns and loss of hair were produced, temporarily, in all animals.

Most conspicuous of the findings in the hearing organ of the rats irradiated on the 11th day in the series reported here were the hemorrhages. They were continuous mostly with the very extensive arachnoidal extravasates, through the inner acoustic meatus. The bleedings were seen together with an engorgement of all vessels in the inner ear and with bleedings around the small vessels of the mesenchymal filling of

the middle ear. Table 2 gives a survey of their location.

The entirely cartilaginous otic capsule was not intrinsically malformed, but its general configuration was altered in 11th- and 12th-day animals by or with the abnormal shape of the head, as described by Hicks (1953). However, in the single ossification center near the orifice of the inner acoustic meatus the formation of new bone probably would have been severely handicapped by suffusion with blood. The same picture was offered in the ossification center in the basisphenoid.

At this period, the cochlear modiolus is formed entirely of connective tissue. It was characterized by engorgement of its vessels as well as by massive or scattered extravasates. Mosher (1938) postulated congestion

TABLE 2.—*Localization of Hemorrhages in the Hearing Organ*

Internal Ear
Internal acoustic meatus
Ossification center near the orifice
Perineural
Endoneural
Cochlea
Connective-tissue modiolus
Engorgement of vessels
Extravasates
Massive
Scattered
Scalae
Tympanic: free (mostly not yet formed)
Middle (cochlear duct): free
Vestibular: blood in portion free of mesenchyma
Vestibulum
Endolymphatic space (entirely formed)
Extravasates, infrequent
Perilymphatic space
Engorgement of vessels
Extravasates, frequent
Middle Ear (Still Filled by Mesenchyma)
Engorgement of mesenchymal vessels
Small perivascular hemorrhages
External Ear
Engorgement of capillaries of the skin
Lateral Sinus
Dilatation by engorgement

of the blood vessels of the cochlea, in addition to the free hemorrhage, as necessary before the hemorrhage can be laid to artificial damage.

Perilymphatic edema was not conspicuous in the cochlea. The membrane of Reissner was hardly ever displaced. At this stage, the scalae, with exception of the cochlear duct, are only partly open. The vestibular scala was farther advanced than the tympanic. The same sequence was observed in the pig by Foley (1931), while in man it is inverted (Alexander, 1930). The free part was filled by blood originating from the mesenchymal vessels.

The vestibular endolymphatic spaces were already entirely free of mesenchyma, while the perilymphatic labyrinth was still more or less occluded. This is a normal finding at this stage. Bleedings were found often perilymphatically, much less frequently within the endolymphatic space. The latter was considerably narrowed by edema in the perilymph. The walls of the endolymphatic sac were in contact; the cochlear aqueduct was narrow and still completely blocked.

The middle ear showed ossicles, nerves, muscles in appropriate developmental phase, with some of the ligaments already outlined within the mesenchyma. Pneumatization was initiated only around the tubal orifice. Small extravasates were found around mesenchymal vessels.

The auditory tube was always patent, containing sometimes blood and debris. The advancing epithelium of its tympanic orifice seemed to be active in the process of pneumatization by compressing the underlying mesenchyma. Rüedi (1937) described enlarging of the air-filled lumen of the middle ear as a mesodermal rather than an epithelial function, while he quoted Eckert-Möbius (1936) as looking for the real determinant of the pneumatization in the bone itself.

The fila of the olfactory nerve offered a normal picture, but large hemorrhages were seen above the cribriform plate between the olfactory tracts.

The structures of the nasal cavity were microscopically normal, including the sensory areas in the upper meatus and in the organ of Jacobson. The vascular plexus of the latter exhibited engorgement of a high degree,

and a similar condition prevailed in the nasolacrimal duct. Secretions in the nasal chambers provoked leucocytic accumulations.

The larynx, which in rat is large and intranarial, was found to be well formed, in its adequate stage of development. Walander (1950) demonstrated in the rat that the vestibular outgrowth is the effect of epithelial activity in the cranial part of the epithelial lamina, a fold in the foregut. The here presented pictures corroborate his statement, according to which the pharyngotracheal duct is patent between the pharynx and trachea throughout embryonal life; however, the intercartilaginous glottis might be closed by approximation of the arytenoids. The edematous folds can be explained as a collateral circulatory phenomenon.

Although the multiple hemorrhages seen in these animals, then, still remain an enigma, their possible origin may be considered. Are they artifacts? In some cases the manner of killing the fetus for pathological study accounted for some small extravasation. The fetus is resistant to anoxia and can live even in fixative for 10 to 20 minutes, during which time anoxic damage to vessels occurs while the heart continues to beat. Slicing the animal rapidly into sections, as a means of preparation, also may induce hemorrhages, but none of these procedures account for some of the blood which had accumulated in most of the places described. Furthermore, the bleedings were largely limited to the near-term animals of the 11th-day radiation experiment. Fetuses examined at earlier intervals did not show these particular hemorrhages. What then is the cause?

Hemorrhage can be an immediate radiation effect or originate later through permanent damage done to the vessels. The finding of blood by Warkany and Schraffenberger (1947) in the vaginal smears of rats following irradiation pointed to an immediate effect. According to Hellman (1952), disturbance of the capillary wall with increase of permeability can result in extravasates, hemoconcentration, and edema.

Primary radiation damage can be excluded here, because at the time of irradiation only the earliest beginnings of the major vessels are present, and they showed no acute impairment. Developmental anatomic abnormalities of the vessels are presently hard to prove, although the large size of some engorged vessels seemed to be disproportionate to what one might have expected from asphyxia. Part of the malformation characterizing the 11th-day animal is an abnormally large choroid plexus of the posterior parts of the lateral ventricles and the adjacent dorsal third ventricle. The subarachnoid space and its vessels consequently are abnormal in distribution. Possibly this situation could lead to hemorrhages in these regions as term approached. The possibility that abnormality of the heart and pulmonary circulation could cause congestion and hemorrhage was considered. In recent experiments (Hicks, 1954), in which the litters were examined in two stages of development after irradiation, no abnormalities of sufficient severity were found, but further analysis is being carried out.

CLINICAL IMPLICATIONS

Interpretation of facts of human developmental pathology by comparison with data as reported here calls for utmost caution. In a correlation of structure and function, the different age scales must be carefully considered. Injury in fetal life was not demonstrated until term; but abnormalities may not become clinically manifest until later in life. In order to bring together multiple defects on a time basis, for each deformity a period has to be assigned at which it presumably developed (Worcester, 1953). A useful approximation was given by Donaldson (1924): "On the assumption that, dating from birth, the life span of the albino rat is three years, then such a rat may be regarded as corresponding to a man of ninety years." The 10½ postfertilization day in the rat corresponds, according to Russell and Russell (1952), to four weeks in human gestation; they emphasized that time intervals of prenatal development are not directly propor-

tional in man and rat. The period chosen for the present report would correspond to two to six weeks in man: the period of "major organogenesis." Actually the "eleven-day" embryo rat is in the 10- to 20-somite stage. In mechanical or acoustic trauma, degeneration of the organ of Corti progresses for long periods after the actual damage is suffered (Lurie, 1942).

Discussion of the age-old question as to whether hemorrhages in embryonic hearing organs form a part of the basic pathology or not has been given elsewhere (Schall, Lurie, and Kelemen, 1951). Congenital syphilis (Kelemen, 1929), quinine poisoning, and rubella of the mother are conditions, among others, in which bleeding constituted the outstanding feature. Berberich (1925) produced these hemorrhages experimentally, duplicating by artificial congestion the pictures seen with birth trauma. Anoxia manifests itself, histologically, in necrosis and hemorrhages. Albrecht (1930) referred to serious consequences of endolymphatic hemorrhage in the vestibulum as against the less harmful localization in the perilymphatic space. In birth trauma he found far-reaching symmetry in size and localization of the bleeding in the right and the left ear. The consequences as to function are different in cases of endoneural, respectively perineural hemorrhage in the internal acoustic meatus; they have been analyzed elsewhere (Kelemen, 1934). Thielemann * (1930) observed fresh blood in the inner ear 60 days after the injury, in the middle ear after 3 months. After a trauma, Barth (1933) found blood in the tympanic cavity after one year.

Bagg (1929) observed after irradiation with x-rays or radium emanation sanguinous extravasations in the embryonic tissues, especially prominent in the head region and in the meninges.

Rubella of the human mother in the first trimester of pregnancy, as cause of damage to the embryo, offers interesting points of comparison. The 10th to 12th prenatal day in rat corresponds to part of the first human trimester, as at this time the rat ear enters the second third of its full developmental circle. In both instances, the end-organs of the inner ear were encountered in a condition corresponding to the fetal age, without morphologically demonstrable changes (Schall, Lurie, and Kelemen, 1951; Lindsay, Caruthers, Hemenway, and Harrison, 1953). In both instances extensive hemorrhages were to be accounted for. In rubella, in spite of normal end-organs, there is sufficient clinical evidence of hearing defects in postnatal life. No such consequences, so far, were demonstrated in rats irradiated as embryos. Accordingly, as long as adequate functional loss is not reported in the rat, the fact stands out that the end-organs of the inner ear were here, too, undamaged; in this case, in close vicinity to most serious intracranial deformities.

It is of interest to remember that in the very first observation, in humans, on damage to the progeny after x-ray irradiation of the pregnant mother Aschenheim (1920) found a boy of 3½ years who was microcephalic, microphthalmic, an imbecile, practically blind, with hardly a rudiment of speech, but in possession of normal hearing. Aschenheim already could look back on a long list of authors from the previous 20 years who, in their animal experiments with irradiation of the pregnant mother, found the brain and eyes of the fetus to be prominently prone to damage. Through all this period, apparently, comparatively little attention was given to the organ of hearing.

Difficulties arise when generalizations are made from animal experiments to human disease. Ingalls (1952) concluded that the same defects may be caused by wholly unrelated physical, chemical, and microbiologic agents and that, as the number of known teratogenic agents rises, the question of specificity seems to be less important than dosage and timing of the agent used. He assumed that any substance that can kill induces abnormality when acting in critical dosage at an appropriate

* Thielemann, M., in discussion on Albrecht, p. 503.

moment of development. Villee (1953) reported the same sort of abnormality occurring as a result of x-rays, heat shocks, or ultraviolet rays if the insults were given at the same time of development. However, it has been shown (Hicks, 1953 and 1954) that similarities between malformations produced by different classes of teratogenic agents acting at the same period of development are relatively superficial, and that the underlying mechanisms are entirely different. Prolonged anoxia and ionizing radiations, for example, attack different elements in fetuses of the same age, and consequent malformations are essentially different. Many other injurious agents have no effect on development, or they may kill the embryo or fetus outright. Radiation selectively damages primitive differentiating nonmitotic embryonal cells; anoxia damages more mature cells and also causes patchy ischemic lesions. Thus the radiation malformations described here are specific for the agent and the species, and generalities are drawn with caution.

Abnormalities in function can be caused by direct damage to the organ itself or can be factors which attack the latter from their environment. Increased vulnerability of the vessels may appear in the fetus at the end of the pregnancy and predispose to birth injuries. In the present case, although the perception apparatus in the inner ear and the conduction apparatus in the middle ear might well remain intact, destruction of the central cochlear and vestibular pathways would be followed by total deafness and by vestibular deficiencies.

Hydrocephalus occurred with high frequency in embryos irradiated at the 11th day. In human pathology, an example of congenital hydrocephalus with ear deformity was furnished by Cantele (1933).

The vascular plexus in the posterior lip of the pharyngeal orifice of the auditory tube seems to have gone unnoticed so far. The tube was found by Tamari (1953) to be always patent in the human embryo. This lip, in children, was described by Reeves and Brill (1954) as being rugged, slightly nodular; the nodularity was caused by the stomata of numerous glands. This nodularity was seen in the present pictures before the mighty layer of glands around the Eustachian orifice developed. Engorgement in the orificial plexus, as described here, might well be a factor in tubal occlusion; as such it merits further elucidation.

Edematous folds of the laryngeal mucous membrane around the basis of the epiglottis, as found here, play an important role in human pathology (Kelemen, 1953) by contributing to the picture of congenital laryngeal stridor.

SUMMARY

Pregnant albino rats were exposed to x-rays at midterm, and their litter examined histologically close to term. The aim was to compare the radiation effect, as shown by malformation and hemorrhage, with similar, previously described human conditions.

The auditory, vestibular, and olfactory end-organs were found to be structurally intact, in appropriate developmental stages, amid extensive hemorrhages in their vicinity and against a background of severe, radiation-induced endocranial deformities.

The teratological damage as encountered here can be typed as an environmental one, namely, not caused by an abnormal condition of the effective end-organ itself.

The functional effect would be, probably, a total loss after destruction in the central pathways. Moreover, it is known that organs appearing intact immediately after an insult may still degenerate, and vascular radiation defect may become manifest later, in the form of a birth injury or during any phase of postnatal life.

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Surgery of the Difficult Septum

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Much has been written about the septum in recent years. Older procedures which failed in certain types of cases have been subjected to various modifications. New methods have been advocated and, while successful in the hands of some, have been found wanting by others. It is quite apparent, therefore, that surgery of the septum is not as simple as it seemed to be in the days when we had only the submucous resection and before the present era of enthusiasm for rhinoplasty. Confusion results from the fact that no one procedure is applicable to all types of cases. Another fact contributing to the lack of unanimity among rhinologists in this matter is the theory held by some that the septum is not the essential factor in the support of the nasal dorsum. Sacrificing all or the greater part of it, therefore, was advocated when necessary to provide an ample airway. This topic has been argued back and forth and, although discredited by most of the recent observers, is still held valid by others. In addition to the above, there is another factor which contributes to the confusion. This has to do with the question of removing septal cartilage and then replacing it, as against procedures which aim to reposition deflected cartilage in situ.

Presented at the Pan-Pacific Surgical Congress, Honolulu, T. H., Oct. 14, 1954.

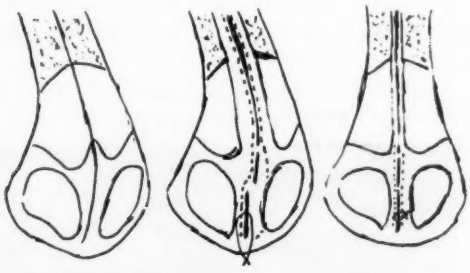


Fig. 1.—Central drawing shows septal cartilage severed in two places to permit repositioning in midline. Broken lines indicate mucosa attached on one side and dissected off the other. Temporary suture drawing lower severed segment of cartilage forward when x-ray film is to be sewn in to splint the septum (Fig. 5).

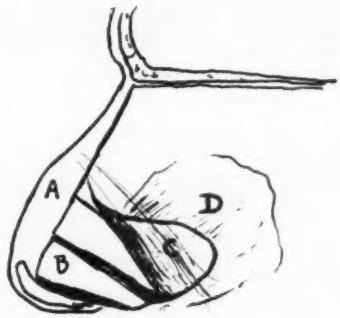


Fig. 2.—*A*, prominence consisting of bone, upper lateral cartilages, and septal cartilage removed to straighten and lower the nasal dorsum. *B*, section of septal cartilage isolated by wedges removed above and below it (solid black). *C*, mucosal flap dissected off the septum on one side. *D*, area of septum removed to correct posterior obstruction.

It is not the purpose of this paper to analyze or discuss the merits of these controversial issues. What concerns us at this moment is the surgical treatment of one certain type of deformity which is not amenable to either the classic submucous resection, the "take out and put back technique," or to simple hacking. We refer to the nose which is scoliotic, long, and humped. The procedure which we will describe is one that we have employed for several years and which has yielded satisfactory results. The objective has been to correct the external deformity, relieve the patient of his nasal obstruction, and still retain most of the septum as a solid median buttress.

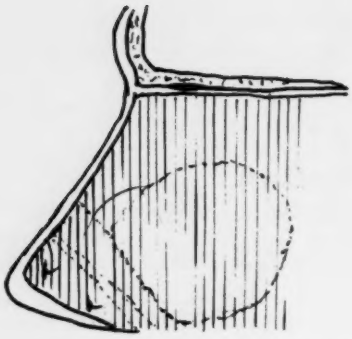


Fig. 3.—The mucosal flap sutured back in position. Broken lines indicate areas removed and cartilage hacked to straighten the curve.

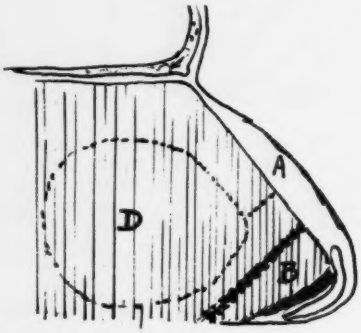


Fig. 4.—View of opposite side, where the mucosa has been left attached.
See Figure 2 for meaning of abbreviations.

The problem presents itself thus. When one removes a fairly prominent hump, he is taking away sections of the nasal bones,

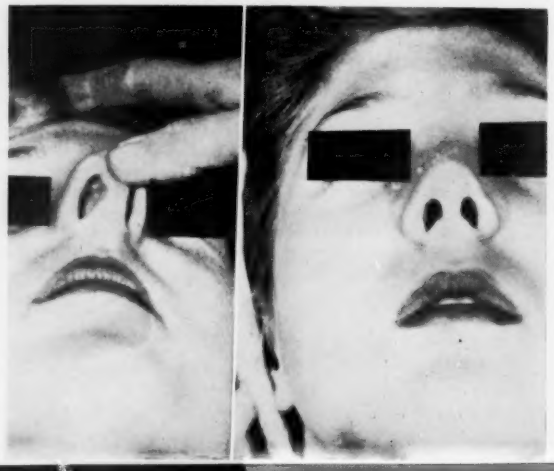
Fig. 5.—Drawing on the left shows x-ray film held by one mattress suture. The film tends to tilt upward. Drawing on the right shows the film brought down to the nasal floor and the tilt overcome by the lower mattress suture. Drawing in the center shows the two strips of film splinting the septum. The dots indicate portions of the film extending into the recess of the lobule above and the floor of the nose below. Packing fills the remainder of the nasal chambers.

Fig. 6.—Front view of patient before (left) and after (right) operation.

the perpendicular plate of the ethmoid, and portions of the upper lateral cartilages, as well as the ventral edge of the septum. As a result, the septum stands isolated, supported posterosuperiorly by its attachment to the sphenoid, superiorly to the frontal bone, and inferoanteriorly to the maxilla. However, it is still curved and frequently presents a crest or ridge projecting into the side opposite the convexity of the cartilage. The important question, then, is how to straighten out the curved septum without sacrificing the cartilage.

In the procedure which we employ, after the hump has been removed and the lower border of the septum resected to shorten the nose, we proceed to detach the mucosa of the septum on one side (usually the convex side) from the exposed lower border all the way up to the ventral border and superoposteriorly far enough to expose the entire curve in the cartilage. Now, one or two narrow triangular segments of cartilage are removed, apices above and bases below, to mobilize the segments and facilitate re-

Fig. 7.—View of septum of patient in Figure 6 before (left) and after (right) operation.



positioning in the midline. In most cases it is necessary at the same time to free the cartilage from its attachment to the vomer. The liberated segments of cartilage are held in place only by the mucosa on the side opposite to the denuded side and are therefore extremely mobile. They have a tendency to flop backward into the nasal cavity and override the bony septum above and behind. This need cause no alarm, however, since they will later be drawn forward into place and secured by the splints, which will be described.

After the segments of septal cartilage have been liberated, one may proceed to remove those portions of the vomer which project into one or the other meatus in the form of a crest or ridge. The perpendicular plate is infracted toward the midline after the osteotomy is performed.

A temporary gauze pack is inserted, and the mucosa which was originally stripped down is reattached to the membranous septum on each side by interrupted nonabsorbable surgical (silk) sutures.

The lower detached segment of the septum is then drawn forward and held by the assistant while two strips of x-ray film are introduced into the nose and sutured into place by two mattress sutures through and through, so as to hold the septum firmly between them. It will be noted in Figure 5 that the strip of film with only one suture through it tends to tilt upward. It is important before passing the second or lower suture through that the film be rotated toward the floor of the nose. Thus the film acts as a splint on both sides of the septum, ensuring healing in the midline. We insert the usual nasal packing external to the x-ray film for additional support.

The packing is left in place for two to four days, and the films are not removed until at least seven days. A copper molded splint is shaped to fit the nose and applied as described in a previous communication.[1] This is worn for six or seven days.

In those cases in which it is not necessary to remove a hump, the procedure is easier because the upper lateral cartilage on the concave side need not be severed from the septum, thus ensuring additional stability while the operation is in progress. In other respects, however, the technique is the same.

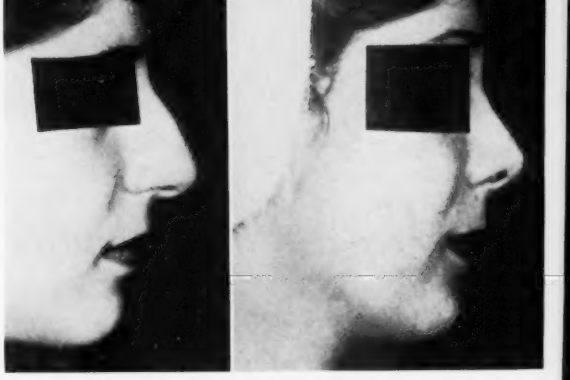


Fig. 8.—Profile view of patient in Figures 6 and 7 before (left) and after (right) operation.

Fig. 9.—Profile view of patient before (left) and after (right) operation.

25 E. Washington St. (2).

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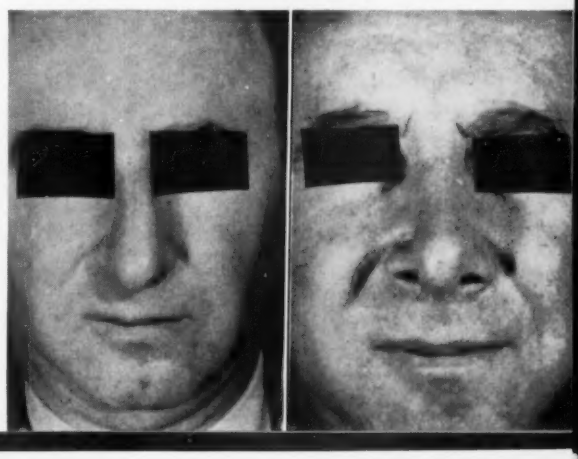
Fig. 10.—Front view of patient in Figure 9 before (left) and after (right) operation.



Heparin in Perceptive Deafness

EMERY I. PICK, M.D., Los Angeles

Regardless whether a hearing defect is a scalar or a nerve type, the etiology of perceptive deafness is dubious in many cases. An etiologic classification might include:

- Congenital anomalies
- Endogenous and exogenous injuries, toxins, acoustic traumas
- Acute and chronic infections
- Tumors
- Psychogenic deafness
- Labyrinth otosclerosis
- Systemic diseases
- Cardiovascular diseases and blood dyscrasias
- Allergic vascular and tissue changes
- Metabolic disorders
- Hormonal, enzyme, vitamin disturbances

Little is known about the etiology of a large group of cases of end-organ deafness, developing around middle age, progressing slowly or rapidly, and, in many instances, preceded or accompanied by tinnitus. It has been tagged as ischemia, but what is behind the ischemia? Vascular spasm? The latter seems to be very doubtful in view of the latest works on arteriolar and precapillary physiology. Is it a deficiency of the hormone, enzyme, vitamin complexes necessary in tissue oxygen supply and in utilization of oxygen by the cell? Or is the etiology an atherosclerosis, which may be considered a disorder of fat metabolism? In this presentation, atherosclerosis has been assumed to be the etiologic factor in a group of patients and the possible prevention on that basis shall be discussed.

In the last few years evidence has been accumulating that atherosclerosis is a disorder of fat metabolism in which plasma lipids infiltrate the vascular wall. The lipids in the serum are carried in the form of lipoproteins of varying molecular size. It has been suggested by Gofman and co-workers that the S_f 12-100 class of lipoproteins are associated with atherosclerosis and are able to induce the lesions in question. All lipoproteins contain lipids like cholesterol, phospholipids, neutral fats and proteins. The varying amount of the components will determine and characterize the different lipoproteins.

The vascular wall may act as a molecular filter and molecular sieving occurs; the S_f 12-100 lipoproteins are sifted out and deposited intramurally. The larger S_f 100-100,000 molecules cannot penetrate the intima at all. During their transport in the serum, the larger lipoprotein molecules are broken down into smaller S_f 4-10 molecules which are able to penetrate into the vessel wall but, as they are able to pass completely through, will not form deposits. This breakdown of the lipoproteins may be due to heparin and is probably one of the several physiologic functions of heparin in the body.

Within several minutes after an intravenous injection of heparin a shift may be observed from the larger to smaller particles with almost complete wipe-out of the larger atherogenic group. In three to four days the larger lipoprotein molecules reaccumulate.

Graham and co-workers first reported improvement in some patients with angina pectoris by using heparin, and Engelberg, applying heparin in coronary and peripheral arterial atherosclerosis, noted that several patients who complained of tinnitus reported a decrease in their tinnitus.

This observation, the effect of heparin on atherogenic lipoproteins, the belief that in a large group of patients with perceptive deafness atherosclerosis is the etiology, and the

heretofore ineffectiveness of any therapy in end-organ deafness initiated the present study. In many cases of this type of perceptive deafness a defective hearing, with or without tinnitus, should be as good an indicator of local anoxia in the inner ear as angina pectoris is in coronary diseases.

Of 40 patients with perceptive deafness, 5 have been excluded—3 because of diabetes and 2 because of syphilis in their youth. The remaining 35 patients, with 70 ears examined, represent 68 ears with defective hearing. The series was initiated in September, 1951.

Of the 35 patients studied 19 had bilateral and 16 had unilateral complaints. However, it was interesting to see that in the unilateral group only two ears had good hearing; all the others showed hearing defects without producing actual complaints. The average age of the male patients was 66 years and of the female patients 59 years; the youngest man was aged 57 and the youngest woman 53. Eighteen of the men and five of the women smoked tobacco. Blood pressures of 160/90 or higher were recorded for 11 of the men and 5 of the women.

In all cases the blood cholesterol ranged between 280 and 350 mg/100 ml. The S_f 12-100 lipoproteins were high. The average clotting time was three minutes. The duration of complaints was from 6 months to 20 years. All patients had had previous therapy for their ear complaints without any benefit.

The bleeding and clotting times were checked at the beginning of treatment, and later, once every month. This proved to be sufficient. The diets of the patients were not altered. Patients with any hemorrhagic conditions were excluded. The audiogram of each patient was checked every month during the course of treatment. With slight corrections we had seven types of audiograms. Type 1, steep high-tone drop, was recorded for six patients. Fourteen patients had audiograms of Type 2, characterized by a gradual high-tone drop. Only one patient had a Type 3 audiogram, with midfield loss. Three patients had Type 4 audiograms, characterized by a flat curve and losses between 40 and 50 db., and six had Type 5 audiograms, with flat curves and losses between 60 and 80 db. Two patients had Type 6 audiograms; in these there was moderate low-tone loss, with hearing best for the 1024 cps frequency and from there on gradual high-tone loss. Three patients had Type 7 audiograms, with a decline for hearing in the 4000 and 8000 cps frequencies. These patients did not complain of hearing impairment; their only complaint was of annoying tinnitus.

The bone conduction did not show any unexpected discrepancies; in most cases it was lower than the air conduction except in Type 4. All patients, before receiving heparin, were placed on placebos for a month. The heparin treatment was started with 100 mg. of heparin intravenously twice weekly. Since we did not notice any ill effects or therapeutic responses, the dose of heparin was increased to 150 mg. and in the last few months to 200 mg. twice weekly. The intravenous route was replaced with subcutaneous and intramuscular injections. In some of the cases we had to revert to intravenous administration because of hematomata occurring even when the injections were given slowly. All patients had previously received a variety of treatments without benefit. No other treatment and medication except barbiturates were allowed during the course of heparin.

Two of the patients stopped treatment after three months. Both had had at the start of heparin treatment tinnitus and an audiogram curve of Type 7. They stopped treatment because tinnitus, which had been their only complaint, improved to the point where it was no longer annoying. The audiograms did not show any change at the cessation of treatment.

Seven patients interrupted their treatment after six months; two moved to other cities, four were restricted by new working conditions, and one became tired of treatment. These seven patients showed no pure tone hearing improvement, but their speech dis-

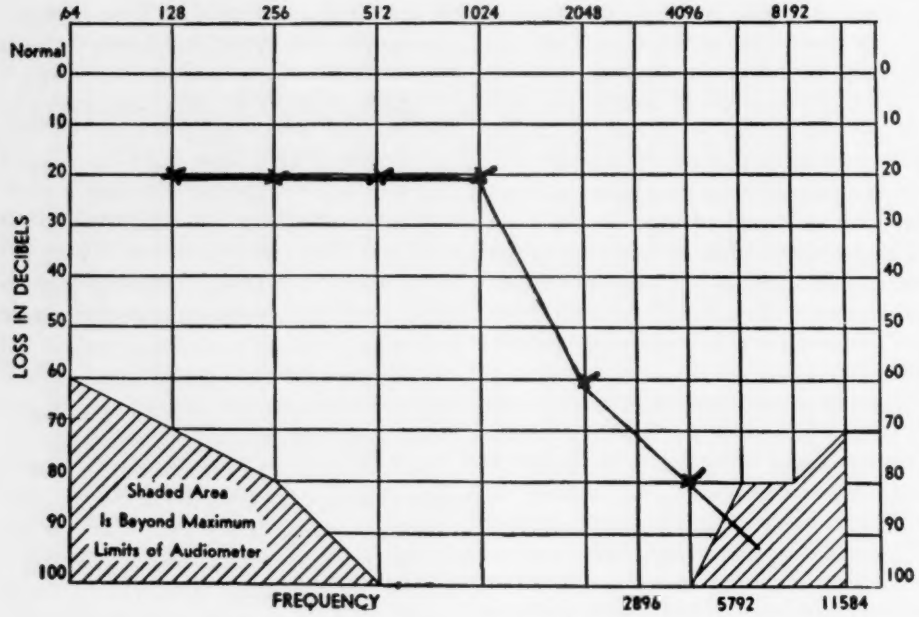


Fig. 1.—Type 1 audiogram—steep high-tone drop.

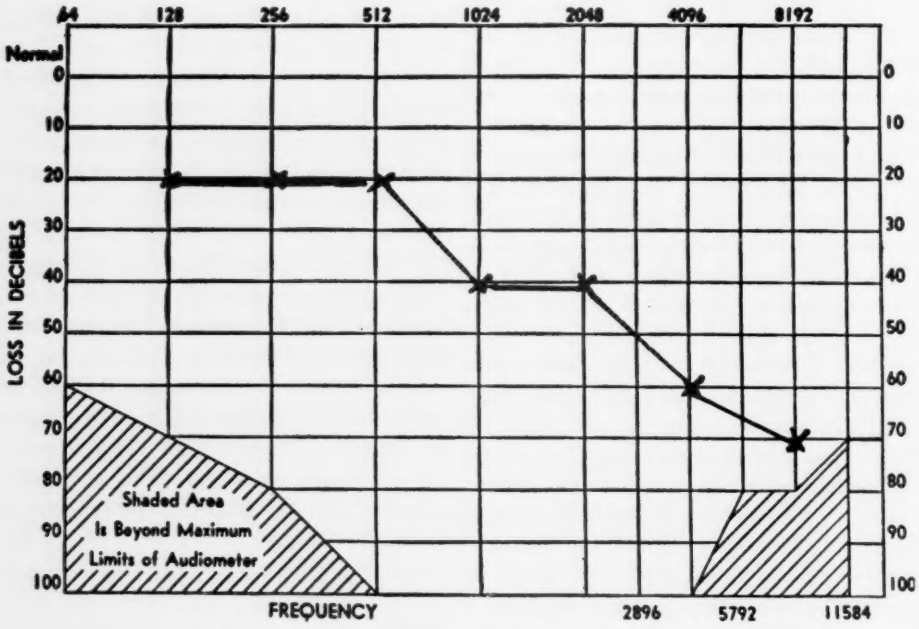


Fig. 2.—Type 2 audiogram—gradual high-tone drop.

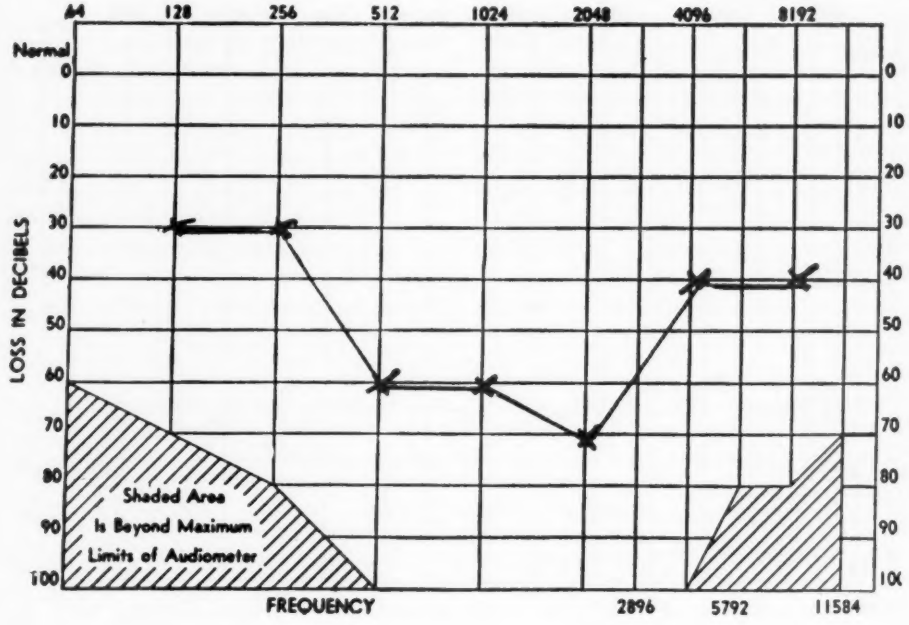


Fig. 3.—Type 3 audiogram—midfield drop.

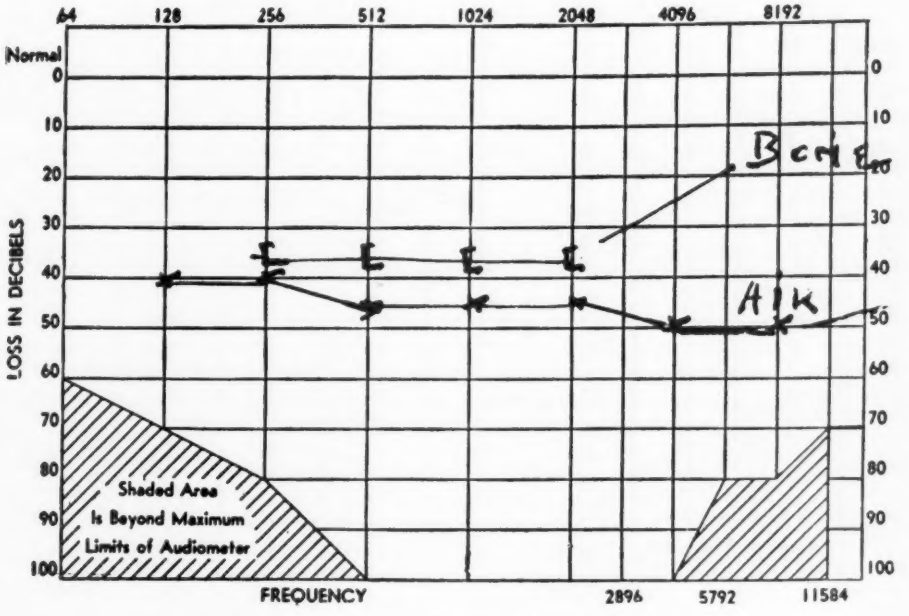


Fig. 4.—Type 4 audiogram—flat curve between 40 and 50 db.

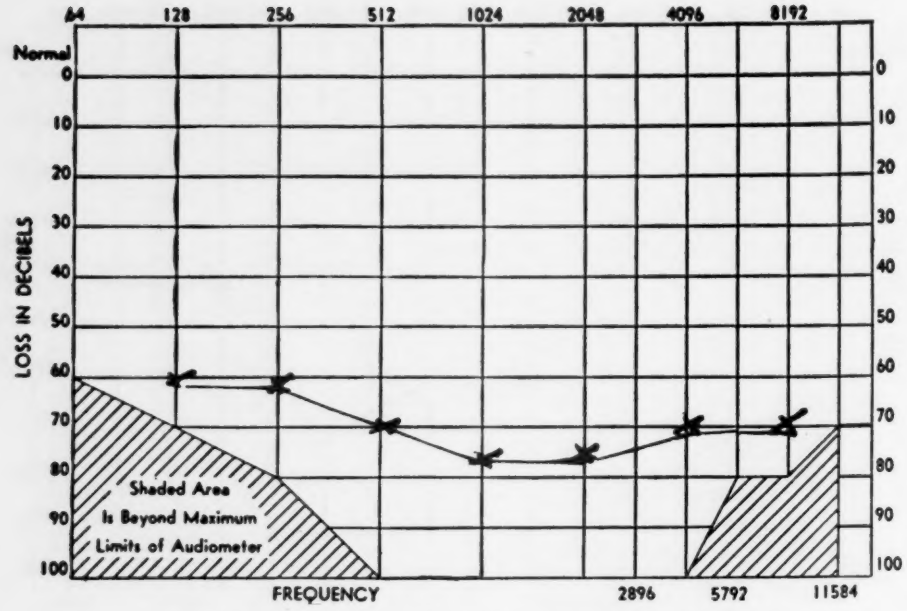


Fig. 5.—Type 5 audiogram—flat curve, loss between 60 and 80 db.

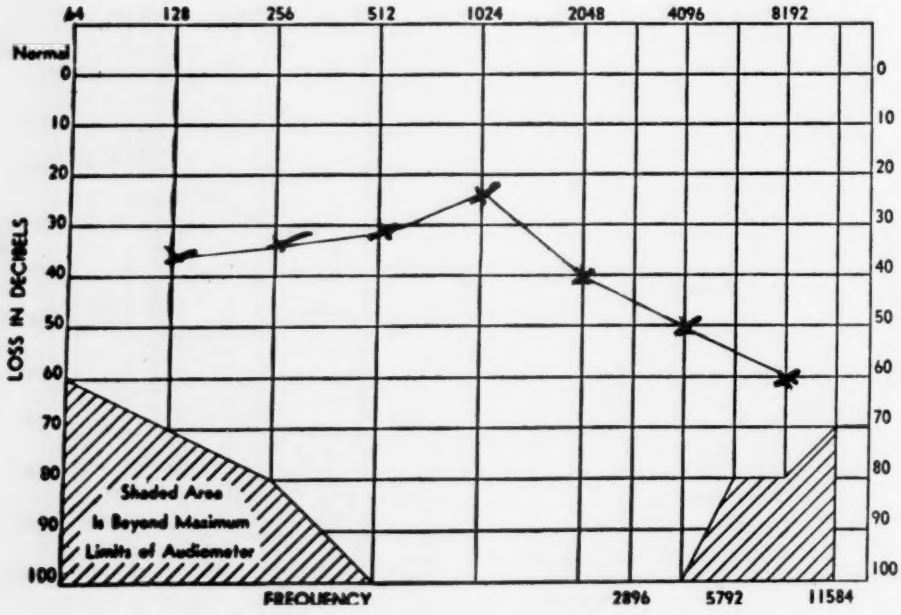


Fig. 6.—Type 6 audiogram—moderate low-tone loss, with hearing best for 1024 cycles and from there on gradual high-tone loss.

crimination and tinnitus were much improved.

Six patients stopped treatment between the 9th and 12th months. In three of these, the audiograms were unchanged, and three others showed an average gain of 10 db. but this was not enough to bring their hearing up to the practical speech level. Speech discrimination and tinnitus improved in all six.

The remaining 20 patients have now been under treatment for from 12 to 20 months. Fourteen of them have had no improvement in their pure tone audiograms; however, their tinnitus was much relieved. Six of them showed a gain of 10 to 15 db., but in only two cases was this enough to bring their hearing up to the practical speech level. In two cases tinnitus disappeared completely; in the remainder it improved.

What is the essential action of heparin? Does it improve the capillary blood supply to the inner ear? Does the heparin prevent a deterioration of atherosclerosis by its action on the plasma lipoproteins? Does the heparin inhibit clotting and sludging of the blood? Does it increase the oxygen utilization of tissues? Does the protein-combining power of the heparin alter the colloid chemistry of the labyrinthine fluids? All these questions can be answered only by further research.

It is apparent that in order for any benefit to be expected the treatment with heparin must be a prolonged one. Even so, only slight, if any, hearing improvement may be expected.

Arrest of the disease and preservation of hearing by preventing further degeneration must be considered a real achievement.

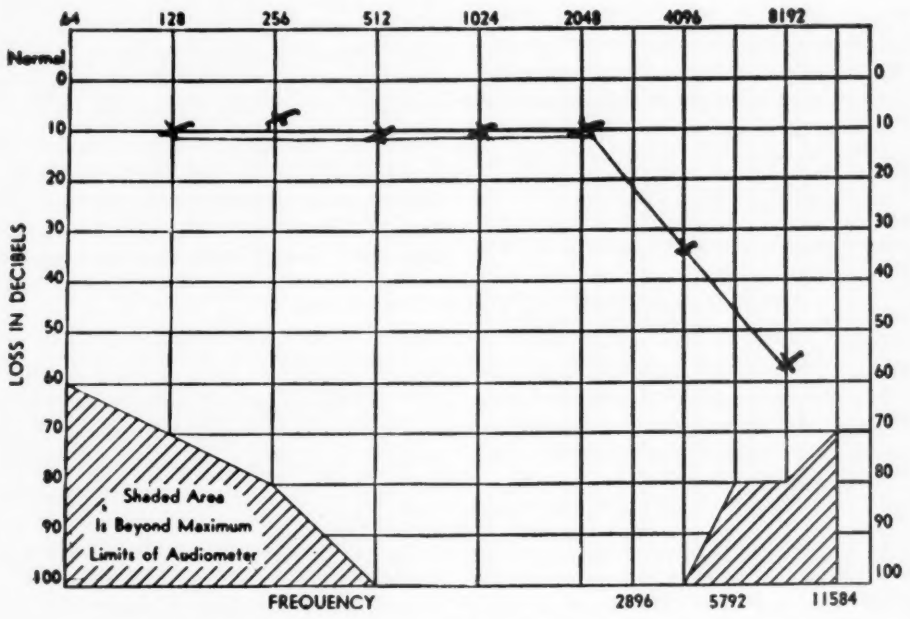


Fig. 7.—Type 7 audiogram—decline in the 4000 and 8000 frequencies.

No further deterioration of hearing was noted in any case during treatment. All patients after a few months of treatment have a feeling of well-being: all have an increased mental alertness and, with it, increased cortical speech perception. The tinnitus stopped completely in two cases. It improved in every case and in the disturbing types it lost its annoying character.

A true indication of the heparin effect on tinnitus is given in the three cases where, repeatedly, shortly after interruption of treat-

ment the tinnitus promptly returned. No toxic reactions, hemorrhage, or other ill effects were noted.

Better results could, no doubt, be obtained if the heparin were given earlier in the disease and in younger patients. In a large measure, the purpose of this presentation is the hope of stimulating interest in the further investigation of the action of heparin in the various progressive diseases of the labyrinth.

SUMMARY

Thirty-five patients with perceptive deafness have been treated with heparin. Improvement of hearing was slight if any. However, the effects of heparin therapy on tinnitus and on other factors influencing hearing were far better. Optimal dosage, application and mechanism of action must be determined by further research.

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Head and Neck Tumors

LT. DANIEL J. FAHEY (MC), U.S.N.R.

INCIDENCE

Head and neck tumors reportedly occur in men 7 to 10 times [1] as frequently as in women. One wonders if this doesn't imply a sex linkage in reference to their etiology. There are two exceptions, which are salivary gland and thyroid tumors. These are even in male and female distribution and may even be slightly more frequent in women.

Head and neck tumors occur mostly in the "cancer age," that is, the middle and later decades. Again salivary and thyroid carcinoma prove exceptions. They occur over a much broader age span.

PATHOLOGY

Ninety-five per cent of these head and neck tumors are squamous carcinoma. This means they usually arise from skin or mucous membranes of the head and neck. Pathologists differentiate squamous and epidermoid carcinoma. The term squamous carcinoma is reserved for these tumors in which pearl formation due to the presence of a keratin-forming element is the predominant feature. Clinically squamous and epidermoid carcinomas are the same; that is, their symptomatology, treatment, and obtainable cure rate are approximately the same.

As Waldeyer's ring of lymphoid tissue is approached in the pharynx there is greater likelihood of primary tumors of unicentric origin occurring. Differentiating these tumors is difficult and this is reflected in the pathologic terms lymphoepithelioma and transitional-cell carcinoma. Although these lymphoid tumors are in a minority, their recognition is important as somewhat better results are expected from radiation therapy than with surgery. Rare adrenocarcinomas arise from the minute glands of oral mucosa and sarcomas from the connective tissue elements in the vicinity.

CLINICAL HISTORY

Autopsy of cases dying with intraoral carcinoma shows that in over 80% * of cases the disease remained limited to head and neck regions. The object of all present-day therapy of head and neck cancer is to institute aggressive intervention at the stage when the tumor is confined to the primary focus or to the primary focus plus regional lymphatics. When tumor has spread beyond these two, no cure is ever obtained.

Salivary and thyroid gland carcinomas are again the capricious ones; they spread by blood stream early and in some cases kill the patient without showing involvement of cervical lymphatics. However, the local lymphatic spread of these salivary and thyroid carcinomas does occur in a significant number of cases. Therefore, despite recognition of the possibility of blood-stream spread, the surgical treatment of the local lymphatics is important in dealing with these lesions.

The main unsettled question is when and where the more extensive removal is required. Progress is being made in increasing the five-year cure rate in many head and neck cancer cases as never before. It is being done by those surgeons who with the aid of modern surgical advances are becoming more and more daring and those who have become very well versed in the clinical history of the various types of head and neck tumors. They are the ones who trap these

Read before the Monthly Staff Conference at the U. S. Naval Hospital, St. Albans.

* References 2 and 3.

tumors, using a superior judgment of when they can cut and when they can melt and blast with radiation.

SYMPTOMATOLOGY AND DIAGNOSIS

Tumors of the head and neck are usually found by three methods:

1. Accidental discovery of a painless sore. A sore found in the mouth which fails to heal with routine medication should be assumed to be cancer until proved otherwise by biopsy. Cancer is the one possibility on the list when an erroneous diagnosis with resultant delay may mean death.

2. The next commonest way they are found is through development of dysphagia, hoarseness, sore throat, dyspnea, or some such complaint. In these instances a tumor may be expected to be found in a relatively obscure recess of the oral cavity.

3. The third and most important way these tumors may manifest themselves is through the appearance of a lump in the neck. The absence of discomfort or dysfunction due to the primary carcinoma is of little significance in the differential diagnosis. Such lumps in adults should be considered cancer until proved otherwise. Hayes Martin dogmatically states, "Asymmetrical enlargement of one or more cervical lymph nodes in the adult is almost always cancerous and usually is due to metastases from a primary lesion in the mouth or pharynx." [4]

The crux of this problem is the failure to recognize the probable cancerous nature of the lump and the failure to recognize that practically all such cancerous lumps are metastatic in nature. The best result of treatment is dependent on the complete diagnosis of primary growths as well as metastases.

It is stated that 60% [4] of cases of carcinoma of the nasopharynx first present themselves with a neck node as the first symptom and 8% [4] of cases of carcinoma of the thyroid do the same while 7.8% [4] of cases of mouth and pharynx cancer first present with a lump in the neck. Cervical metastases are the first symptoms of lesions below the clavicle in only 0.6% [4] of cases of lumps in the neck.

In cases with lumps in the neck 90% [4] of the primary tumors can be located with a thorough examination. With location of the primary tumor and biopsy of it, a coexisting lump in the neck is assumed to be metastatic disease. In a certain percentage of cases the primary tumor will not be located. In these cases a needle aspiration biopsy should be performed. Lymph nodes should never be excised, as such removal is considered by authorities as a most reprehensible surgical procedure. They have found that it invariably lessens the chances of cure. The effect of such an excision produces scarring, which prevents or precludes clean surgical neck dissection. There are exceptions to this rule, when a primary tumor has not been located after a very thorough search and after adequate needle biopsy has failed to give a diagnosis. At such an excision procedure, however, a frozen section should be made on opening the neck, and one should be prepared to do a complete neck dissection.

Hayes Martin reported a series of 163 cases [4] presenting with neck lumps. In only 16 had the primary tumor been established by biopsy. Of these 163 cases, 61 (37%) had had a lymph node removed. In 40 of these (65%) the perfectly obvious primary lesion was discovered on the first visit.

Excising a node to establish a diagnosis is only of academic interest. Before anyone prepares to open the neck for a lump he should stop and ask himself, "What would I do if the diagnosis is carcinoma?" The answer is (1) a more thorough examination in an attempt to locate the primary tumor, (2) aspiration biopsy, (3) x-rays, etc., and (4) referral of the patient if the physician himself is not fully capable of handling the cancer cases correctly. All of these should be done before any removal of the lymph node. In the examination of the patient attention should be given to the mouth, nose, nasopharynx, hypopharynx, larynx, thyroid, salivary glands, and scalp. If these are all negative, an aspiration biopsy should be done. Repeated direct examination of the larynx and esophagus should be done. Fluoroscopy

and x-rays of the lungs and gastrointestinal tract together with pyelograms should be obtained. If no primary tumor is found, a neck dissection is warranted and justifiable for the removal of carcinoma. With careful follow-up on these cases it is most unusual for the primary tumor not to show up eventually. Some have reportedly done so up to four or more years after a neck metastasis.

The Memorial Hospital group advocates radiation if the single metastatic node is small enough for a 3 cm. x-ray port to cover it.[4] The arbitrary diagnosis of branchiogenic carcinoma should never be made on these cases as it precludes a careful scrutinizing follow-up for the primary lesion, which is very likely to eventually show up.

THERAPY

Leaders in this field at present advocate the following therapy. Local excision is done for very early intraoral lesions. With more advanced lesions with and without cervical metastases a combined resection of the lesion together with the mandible and neck dissection (termed a commando procedure) is used. A series of 200 such cases [4] were reported from the Memorial Hospital in New York in which there was no gross evidence of metastatic disease. Microscopic evidence of metastases was present in 17% of these. The five-year cure rate with this approach is over 40%, which is the best established thus far.

The therapy for nasopharyngeal, tonsillar, and hypopharyngeal lesions is radiation for the primary lesion and also for the metastases, unless the primary growth is controlled, and then neck dissection is considered. Intracavitary radium or radon is used, as well as fractionated multiple-port roentgen therapy.

The therapy for early intrinsic larynx lesions is laryngofissure with excision of the disease when limited to one cord. If the disease is more advanced, a total laryngectomy is done. For extrinsic lesions a combined neck dissection with total laryngectomy is performed. In some centers it is felt that advanced intrinsic lesions, that is, supraglottic extension, require the combined procedure also, regardless whether gross evidence of metastases is present or not.†

Some salivary gland and thyroid carcinomas are also treated with a combined resection and neck dissection without gross nodes being present. It has been stated that in 50% of cases, with papillary nodular carcinoma of the thyroid, without metastases grossly, microscopic evidence of spread is found.

Speaking of thyroids, general practitioners and internists are called upon to handle thyroid nodules very frequently. In 10%-22% [5] of cases with a solitary nodule in the thyroid, carcinoma is found. Such solitary nodules should obviously be removed widely, in 50% of cases with papillary nodular carcinoma, a neck dissection together with wide excision of the nodule should be carried out.

I would like to reiterate that even though the treatment of head and neck cancer has its disappointments and failures, the results being obtained today are definitely being improved over the past, as much as or more so than in other areas of the body. The main reason for this I believe is the fact that this disease remains a local one.

In conclusion I would like to say that the treatment of these cases of head and neck tumors requires a variety of skills which today are in various specialties. This accounts for the reason why these lesions are in a medical no-man's land. The otolaryngologist skilled in examination of the orifices of the head and neck may be able to discover primary tumors. His ability to cope with the case, then, by neck dissection and jaw resection, etc., is frequently lacking. Such an ear, nose, and throat man without much training can contribute little to the actual therapy though he may make the diagnosis. Many radiologists unhesitantly accept responsibility for therapy of intraoral carcinoma, and yet it is unusual for the average radiologist to be capable of examining or even willing to examine the pharynx, larynx, pyriform sinuses, or base of the tongue with a view toward appraising the extent and

† References 5 and 6.

limitations of the primary tumor. Cervical metastases, the commonest complication of intraoral cancer, are more efficiently treated by surgery than by x-ray, and even though the radiologist may sterilize the primary lesion, he has to bow out and turn the patient over to someone else to care for this eventually. The general surgeon or his colleague the plastic surgeon may be qualified technically to perform a neck dissection but again many times lacks the ability or willingness to thoroughly examine the oral cavity to find the primary tumor. Further on the fringe of those toying with the problem are the oral surgeons and oral pathologists. They are trained in dental schools, where no opportunity exists for gaining familiarity with general surgical techniques. The only answer to this situation, as I see it, is that the persons who wish to handle these problems should first thoroughly understand the problem and then equip themselves with whatever training they lack. Each and every one of these fields has many advantageous skills, the understanding and use of the sum total of which are needed to handle these cases, if not alone, in cooperation with those fields which make up what is lacking.

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The Nasal Aponeurosis and Its Role in Rhinoplasty

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and
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Much has been written about rhinoplasty since Joseph's monumental work.[1] Again and again new techniques have been described for shortening of the nose and the prevention of postoperative drooping of the tip. Because of the frequency of such reports and the fact that the proposed newer techniques were frequently offered without any basic reasons given for the changes, it appeared to us that some common ground could be found for explaining the advantages and disadvantages for the procedures advocated.

For clinical reasons, it appeared to us that a study of the aponeurosis (fascia) of the nose might throw additional light on the subject, and lead to some standardized surgical procedure for shortening of the nose and a reasonable assurance that the lobule would remain in its elevated position long after operation.

ANATOMY AND EMBRYOLOGY

Only that part of the nasal aponeurosis which is attached to the pyriform margin of the nasal osseous aperture, on the one hand, and to the upper and lower lateral cartilages, on the other, will be discussed in this paper.

Embryologically the upper and lower lateral cartilages do not make their appearance, as such, until the sixth month of fetal life.* A fibrous sheet growing in from the pyriform margin divides the original single cartilaginous nasal capsule into several divisions; these are the upper cartilaginous vault with its contiguous septum, the two lower lateral cartilaginous arches, and, finally, the multiple sesamoid cartilages.

This nasal fibrous sheet (aponeurosis) occupies an irregular space, bounded dorsally by the pyriform margin of the maxilla, cephalically by the caudal margin of the nasal process of the maxillae and caudal margin of the nasal bones, ventrally by the dorsal margin of the upper lateral cartilage and that part of the ventral margin of the septum which lies between the caudal margin of the upper lateral cartilage and the cephalic margin of the lower lateral cartilage, and caudally by the cephalic margin of the lateral and mesial crura of the lower lateral cartilages (Figs. 1 to 3).

FUNCTION OF THE NASAL FASCIA

The complex attachments of this fibrous sheet serve a dual purpose. The first is that the lobule can be moved in all directions and still maintain its recoil action. The second is that it serves as a support for the nasal tip.

MECHANISM OF MOBILITY AND SUPPORT OF LOBULE

The following is offered as an explanation of how the nasal fascia supports the lobule and at the same time allows for its mobility.

We consider the skeleton of the lobule to be dynamically a single arch, instead of two anatomical arches united in the midline, because, in fact, during motion of the lobule the two arches act as a single unit rather than

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* References 2 and 3.

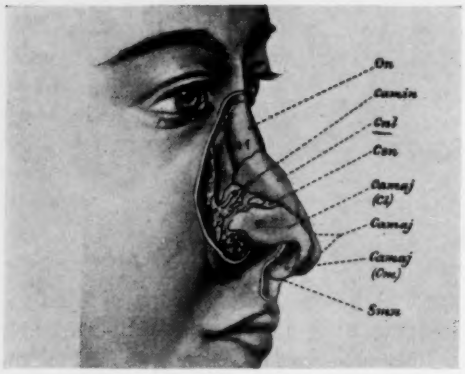

Fig. 1.—Cartilages of external nose with nasal aponeurosis (front view). (Reproduced from Schaeffer, J. P.: The Nose and Olfactory Organ, Blakiston Company [division of McGraw-Hill Book Company, Inc.].)

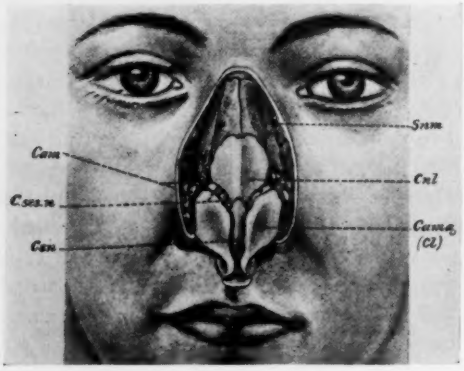

Fig. 2.—Cartilages and aponeurosis of external nose (lateral view). (Reproduced from Schaeffer, J. P.: The Nose and Olfactory Organ, Blakiston Company [division of McGraw-Hill Book Company, Inc.].)

Fig. 3.—Low-power study of section taken through limen vestibular of nose. Upper and lower lateral cartilages surrounded by nasal aponeurosis and overlying mucosa.

as two independent units. We believe further that the architecture of the lobule and its mode of action are similar to that of a "moat bridge." Reference to important parts of such a bridge discloses that it is suspended by chains attached to the castle wall. The chains attached to the flanks of the bridge keep it drawn up or dipped, depending on whether there is no slack or much slack in the chains. In a similar fashion the nasal fascia, anchored in the bony pyriform margin of the maxilla, spreads out fan-like and attaches itself to the skeleton of the lobule, i. e., the lower lateral cartilages (Fig. 4). The lobule is thus supported by the nasal fascia. Any relaxation or excessive lengthening of the fascia gives rise to a "dipping" or drooping of the lobule

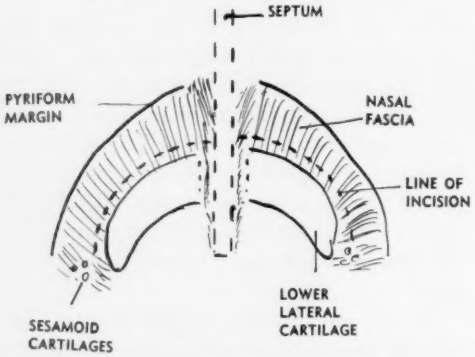

Fig. 4.—Diagrammatic drawing of the attachment of the nasal aponeurosis from pyriform margin to lower lateral cartilages.

of the nose. One further point with reference to a "moat bridge": hinge joints, at either flank, adjacent to the castle wall serve as the pivoting points for motion of the bridge. The pivoting points of the lobule are likewise at the flanks. The dorsal ends of the lateral crura are close to the sesamoid cartilages near the pyriform margin of the maxilla, with some nasal fascia interposed. This peculiar anatomical arrangement serves as an unusual type of joint in that during motion of the lobule the lateral crura slide over the sesamoid cartilages, simulating the hinge-joint action of a "moat bridge." This can be easily demonstrated by shoving the lobule cephalically and observing it jump back into place, or pulling the tip down and watching it rise to its original position.

NEED FOR LOBULE MOBILITY

Mobility of the lobule is necessary for a variety of reasons: 1. When trauma is applied to the tip, the lobule must be mobile enough to ride with the blow and yet be able to recoil to its original position. 2. The same mechanism comes into play in the act of grimacing. 3. During growth of the nose the quadrilateral cartilage of the septum grows in a ventrocaudal direction, carrying the nasal fascia along with it. The elasticity of the fibers of the nasal fascia in addition to their lengthening allows for mobility of the lobule and its displacement ventrocaudally during this period.

CAUSES FOR DEPENDENT LOBULES—FAILURES IN SUPPORT

As long as the quadrilateral cartilage remains in the midline in the vomerine groove and is not hypertrophied, the nasal fascia is not unduly stretched. However, if the quadrilateral cartilage is too long or if the caudal margin of the septum is twisted or displaced from the midline, the facial fibers are subjected to unusual stress, causing them to gradually lose their tensile strength, with consequent drooping of the tip. These conditions can arise during growth of the nose or as a sequela to trauma.

If the upper lateral cartilages grow very long and project deep into the nasal vestibule, they may cause an undue stretching of that portion of the aponeurosis which is attached to the caudal border of the upper lateral cartilage and to the cephalic margin of the lower lateral cartilage in the region of the limen vestibular. This stretched fascia over many years could be an important factor in the production of a dependent tip later in life.

Finally, unusual hypertrophy of the fascia could give rise to a long nose with a dependent lobule.

It can be seen from the above that this fascia is being subjected to some form of stretching all during life, whether from growth of the nose, trauma, grimacing, etc., or just less of elasticity due to advancing age.

We are all aware of the fact that with advancing age most nasal tips become dependent. We have had difficulty, in the past, finding a logical explanation for this phenomenon.

Most of us have felt that it was due to stretching of the skin. This is hardly likely. If we stop to consider that relaxation of the face with the onset of jowls starts about the 40th year and continues to increase from that time on, we are immediately struck by the similarity of the time of onset of dependent nasal tips. The nose starts to droop, as a rule, near the 50th year, and it becomes more evident as time goes on. We believe that it can be logically explained on the basis of relaxation and stretching of the nasal fascia, the supporting structure for the lobule, and the mechanism is similar to the stretching of the fascia of the face leading to jowls.

SURGERY OF THE NASAL FASCIA AND SHORTENING OF THE NOSE

In order to shorten the nose, the lobule must be converted into a mobile flap, attached at its flanks, so that it can be rotated cephalically. This is accomplished by severing the attachments of the nasal fascia to the cephalic margins of the lower lateral cartilages, i. e., the skeleton of the lobule.

The limen vestibular incision cuts across the long axis of the fibers of the nasal fascia that are attached to the cephalic margin of the lateral crura. In making the limen vestibular incision, it is therefore necessary to start close to the pyriform margin of the maxilla and swing mesially over the septum to meet the limen vestibular incision of the opposite side. If the incision does not go close to the flanks, there could be many stretched fascial fibers which remain uncut. It is true that those aponeurotic fibers that are cut across allow for their eventual shortening by imbrication and with healing keep the tip up. However, the untouched stretched fascial fibers in the flanks cannot be imbricated and therefore do not aid in holding up the tip. This could lead to an undesirable postoperative drooping of the tip (Fig. 4). Finally, the transfixion incision cuts the

attachments of the nasal fascia to the ventral and caudal margins of the septum and the cephalic margin of the mesial crura.

As the result of these incisions, a mobile flap consisting of the lower cartilaginous vault attached only at its flanks is fashioned and the lobule can then be rotated as far, cephalically, as is necessary, using the simulated hinge joints as pivots of rotation.

With the lobule held in the newly desired cephalic position, any excess or redundancy of the septum or upper lateral cartilage is then removed.

In order to maintain the lobule in its newly desired position, it is necessary to strap the lobule at the end of the operation so that the cut fascial fibers are overlapped—this overlapping extends from one hinge joint to the fellow of the opposite site. An adhesive dressing maintained for seven days is sufficient to accomplish this.

After the lobule is fixed in its new cephalic position, it still remains mobile. This is due to the fact that, although the aponeurotic fibers are cut across, the "hinge joints" remain free to function. This is important physiologically.

In passing, one point is made with reference to the use of orthopedic sutures (from columella to septum) to raise the tip of the nose. The reason that this technique fails is that the use of such sutures only succeeds in raising the mesial crura, while the remainder of the arch is not rotated cephalically. When the orthopedic sutures absorb or are removed, the tip immediately falls.

SUMMARY

The nasal aponeurosis is described in minutia, and its physiological action with reference to the lobule is discussed.

The main support of the lobule and its mobility results from the nasal fascia's peculiar attachment to the skeleton of the lobule, i. e., lower lateral cartilages. It is suggested that the lobule is attached to the nose in the same fashion that a "moat bridge" is attached to the side of a castle; further, that the lobule's mobility depends principally on its hinged-joint-like action similar to that of a moat bridge. Dependent tips arising in old age are explained on the basis of relaxation and stretching of the nasal aponeurosis.

For proper shortening of the nose and the permanent maintenance of the lobule in its new cephalic position, due consideration must be given to the length of the incision in the limen vestibular. It should start at or adjacent to the maxillary pyriform margin on one side and extend to the same point in the opposite side, thereby cutting across the long axis of the nasal fibers in this area. This, in combination with the transfixion incision, separates the septum and upper lateral cartilages from the lobule, converting the latter into a mobile flap which can be rotated cephalically, imbricating the cut fibers of the aponeurosis. Any redundancy of septum or upper lateral cartilage is then removed. Simple adhesive strapping for seven days is used to maintain the new position.

If the proposed new concept is borne in mind, better immediate and postoperative rhinoplastic results should be obtained, especially with respect to shortening of the nose and the maintenance of the lobule in its new cephalic position.

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Practical Office Audiology

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This paper is intended for the practicing otolaryngologist. Its only purpose is to describe in a practical manner some of the methods of office audiology that I use and have found to be helpful and reliable.

A systematic and orderly method of examination is extremely convenient in our effort to arrive at an accurate diagnosis, establish a prognosis, and advise the correct treatment to the deafened and hard of hearing adult. Preschool children with auditory disorders require special techniques, which will not be discussed here. The routine for adults is as follows:

1. History
2. Otolaryngological examination
3. Voice and tuning-fork tests
4. Pure-tone audiometry
5. Speech audiometry
6. Vestibular tests

HISTORY

The history is an essential preliminary approach of the utmost importance, because, even if it is not conclusive by itself, in many cases it will point to the etiology of the hearing loss and be a valuable clue in the diagnosis. For instance, a hearing loss which develops several months after dihydrostreptomycin therapy suggests that drug to be the cause. A hearing loss that came on insidiously, is progressive, with better hearing in noisy places, positive family history of deafness, and negative history of ear disease points strongly to otosclerosis. A sudden single attack of vertigo, deafness, and tinnitus in an older person with a previously normal ear suggests a vascular accident. A patient having repeated episodes of vertigo, tinnitus, and deafness with a sense of fullness in the ear points to a case of labyrinthine hydrops. A hearing loss which follows a long history of ear discharge without other symptomatology points to an adhesive otitis media. A patient having had mumps and later on a profound unilateral deafness suggests mumps to be the cause. These examples are given to show that, with the data obtained from a carefully taken history, the otolaryngological examination is approached advantageousiy.

OTOLARYNGOLOGICAL EXAMINATION

The otolaryngological examination should be complete. We wish to insist on three topics. First, examination of the nasopharynx. We think that it should be done routinely with the nasopharyngeal mirror, because it is well tolerated by most patients if taught how to breathe properly. If further inspection and details seem to be wanted, a nasopharyngoscope is useful. Second, if the examiner finds a pale nasal mucosa, watery discharge, and congested turbinates or a central tympanic perforation with watery mucus or thick discharge, the possibility of allergy should be contemplated. In this respect one should remember that the history, skin testing, and finding of eosinophiles are more or less presumptive evidence, but the conclusive proof will be the therapeutic test correctly approached. Finally, to test the permeability of the Eustachian tubes, the use of a catheter and a diagnostic tube to hear the sounds produced by inflation is a good procedure, although we think that it should not be used routinely. As for nasopharyngoscopy, in our experience, no matter how well anesthetized is the nose of the patient and how gentle and careful is the examiner, many patients will complain aloud and those

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who do not will tell us that they find the whole procedure quite disagreeable. We prefer to inflate the tubes by passing compressed air, from our air supply, through a De Vilbiss nebulizer, No. 180, which contains a small quantity of chlorobutanol (Chloretone) (Park, Davis & Company) inserted in the nose. The patient says loudly and clearly: K,K,K,K. At the same time the tympanic membrane is visualized with an ear speculum, preferably with magnification, observing its mobility. If the deafness of the patient is suspected to be due to occlusion of the tubes, we take an audiogram before as well as after inflation, and the two air curves are compared.

VOICE AND TUNING-FORK TESTS

The voice test is convenient because no equipment is needed. We always carry our voice with us and the patient his hand to mask. It gives an idea of the hearing loss. It is better to know the normal voice threshold in the examining room. It can be easily found by testing 10 or 15 normal hearing persons and obtaining an average, which can be recorded with a sign, such as a line, drawn on the floor. The findings should be recorded as the ophthalmologist does: 20/20 for normal vision. The denominator will correspond to the normal in the room. We prefer the whispered voice, after expiration, with residual air, which is around the 3000 frequency. The normal threshold for the whispered voice in our room is 8 ft. There is not enough space available for the normal threshold for the spoken voice in most offices.

We do this test as follows: The patient turns the ear to be examined toward the tester and the opposite ear toward the wall. His eyes should be closed to avoid lipreading. The ear not tested should be preferably masked. We use a masking apparatus given to us by Dr. Val Jordan, of Cleveland, which we found to be most satisfactory for the voice as well as the tuning-fork tests. However, a good masking effect is obtained by having the patient rub his ear with the palm of his open hand, which is better than to have the ear canal stopped with a finger tip, which would remove only about 30 db. We start close to our patient with high-pitch words because the healthy ears hear them better, and continue with low-pitch words. When the patient repeats correctly two words, we move one step backward and repeat the test. Each step backward is equivalent to about 2 ft. If the patient is unable to hear the whispered voice even at 1 ft., the spoken voice is used exactly in the same way and pertinent annotations are made on the chart.

If by the voice test it is found that the patient has enough hearing loss to be handicapped, we try to have an idea of his discrimination. An old-fashioned, commercially available conversation tube, about 3 ft. long, with hard rubber ear and mouth piece, is used to give to the patient's ears words, mostly taken from the PB-50 word list. This test usually gives a pretty good idea of the ability of the patient to make fine discriminations. If there is good intelligibility and, consequently, correct repetition of the administered words, most probably we are dealing with a conductive loss; and, vice versa, if there is poor discrimination and poor repetition of words, most likely it is a case of a perceptive nature.

The next step is to make the differentiation between the two great types of deafness: conductive and perceptive. This should always be done with what we consider to be still the best means: the tuning forks. They will also serve to check the pure-tone audiometry, and when there is great discrepancy, if the results are inconsistent, most likely the error will be audiometric, because the audiometer, being a rather complicated electronic device, occasionally goes out of order. Perhaps another good point of the forks is the fact that most of us do our own tuning-fork tests but have to count on another person to do the audiometry. Our forks are made of a magnesium alloy, and we feel that they are easy to handle, convenient, and accurate enough for air and bone conduction.

It is well known that the young adult ear can hear sounds between 16 d. v. and around 20,000 d. v. That means low, medium, and high pitch sounds. The 16 d. v. fork is more felt than heard, so we start testing with the

32 d. v. fork by air, which will be the lower limit. If sounds are heard, most probably the patient has not a conductive deafness. One must be very careful to present the flat surface of the fork at a fixed distance to the external meatus of about ½ in. to avoid a "silent zone." One must avoid touching the pinna or the hairs. One must be very careful to damp the forks to avoid overtones. The 64 d. v. fork can be very misleading, because, if not damped, it will produce high-pitch overtones. We test by air conduction with the 32, 64, 128, 256, 512, 1024, 2048, and 4096 d. v. forks. If our higher pitched forks are heard, most probably the patient has not a perceptive loss. To test the upper limit by air, we use the audiometer. Most of them go as high as 12,000 cycles, which is the upper limit in most middle-aged adults. In general, low-pitch forks are not heard in conductive losses and high-pitch forks are not heard in perceptive losses, although there are exceptions. For example, secretory otitis with the middle ear completely filled with fluid, although a pure conductive loss, produces a greater impairment for high-pitched tones, while labyrinthine hydrops, though a pure perceptive loss, produces a greater impairment for low-pitch tones. Afterward we compare the threshold acuity for air and bone conduction by means of the Rinne test. We use the 256, 512, and 1024 d. v. forks. The vibrating fork is placed alternately in front of the meatus and against the mastoid, establishing whether the acuity is better by air (Rinne positive) or by bone (Rinne negative) or approximately equal (Rinne equal). We feel that this method is accurate enough and simpler than others requiring stop watches, standard blows, etc. We look for fine details of the hearing loss in the audiometric studies.

The bone conduction is tested afterward by means of the Schwabach test with the 256, 512, 1024, and 2048 d. v. forks. It is important to place the fork in the optimum place on the mastoid bone, which should be found by trial, and exert intermittent pressure by placing the fork alternately on the mastoid bone to avoid fatigue. Again, we feel that comparing the patient's threshold with that of the examiner (assuming the examiner's is normal) is accurate enough and simpler than other methods. Care must be taken with the 2048 d. v. fork so it will really be heard by bone and not by air.

Finally, the Weber test is done with the 256 and 512 d. v. forks, placing them on the vertex of the skull and in the midline and finding in which ear the fork sounds louder, to which ear it lateralizes. In some cases the patient is instructed to place his finger tip in his ear canal alternately while the Weber is being done. In this fashion many times he is able to realize what we mean by asking him in which ear the fork sounds louder.

Negative Rinne tests indicate conductive loss. Positive Rinne tests indicate perceptive loss, although if the fork is heard about twice as much by air as by bone, the positive Rinne means normal hearing. Therefore, the Rinne test should be complemented with the Schwabach test, because an accurate estimation of the bone threshold provides a simple method of assessing nerve function, and this is well accomplished by the Schwabach test. In general, in conductive deafness the Schwabach is prolonged because the examiner's ears are not masked while the patient has a soundproof room of his own. In perceptive deafness it is shortened.

In a progressive conductive loss the Rinne test, being first equal, becomes negative later. If the 256 d. v. fork is negative and the 512 and 1024 d. v. are positive, probably there is a 20 to 30 db. conductive loss. If the 256 and 512 d. v. are negative and the 1024 d. v. is positive, there should be about a 40 db. conductive loss, and if all three forks are negative, the loss should be around 50 to 60 db., which is the maximum possible pure conductive loss.

The Weber test is of limited value in clinical testing because some patients do not seem to be able to lateralize when the tuning fork is placed on the vertex. In general, it lateralizes to the bad ear in conductive deafness (best bone conduction) and to the good ear in perceptive losses. In many cases after

fenestration surgery, its value seems to be great because with a simple method we can test the cochlear response. Usually it lateralizes to the ear operated on and if it does so for a time and afterward changes to the ear not operated on, it may mean serious labyrinthitis in the cochlea.

Sound applied to the mastoid bone can go through the skull and be heard by the opposite ear, losing about 10 db. That means that in some cases for accuracy it is necessary to use appropriate masking. Masking should be used when there is around a 40 db. difference between one ear and the other or when the patient lateralizes the sound to the ear not being tested. When there is a very great difference between the two ears, complete masking may not be possible, for example, in advanced cases of unilateral acoustic neurinoma.

Next we test for diplacusis, which is of great diagnostic value in cases of labyrinthine hydrops. Diplacusis is seen more frequently for the lower frequencies. When the fork is switched quickly from one ear to the other, some patients notice a higher pitch (not intensity) in the impaired ear as compared with the normal. Lately we have been testing for diplacusis by using two identical forks, thus avoiding the quick changing of the forks.

If the patient complains of tinnitus, we try to match it with the vibrating forks, and we record the frequency on the chart. As results of the tuning-fork tests are recorded as normal, slightly shortened or prolonged, moderately shortened or prolonged, much suggested by Dr. George Shambaugh, the shortened or prolonged, and not heard. This gives some idea of the "quantity of the loss," although tuning-fork tests are essentially "qualitative tests."

In general, in conductive losses low-pitch forks are not heard by air; therefore, there is an elevation of the lower limit, Rinne tests are negative, the Schwabach prolonged, and the Weber lateralizes to the worse ear. On the contrary, in perceptive losses, high-pitch forks are not heard by air; the upper limit is thus lowered, Rinne tests are positive, Schwabach tests are shortened, and the Weber lateralizes to the better ear.

PURE-TONE AUDIOMETRY

In regard to the testing room, our ideas are that a soundproof room is neither practical nor necessary for clinical testing, but every practicing otolaryngologist who is interested in hearing disorders should have sound-treated rooms. Our set-up is practical and inexpensive. It consists of two rooms: one smaller, 9 by 4 ft., the control room, and one larger, the examining room, which is 9 by 9 ft. Between the two there is a one-way mirror, which permits looking into the examining room from the control room only. It is a room built within a room, with the floor of the inner room supported by sand. It could have been supported by rubber or cork. The walls and ceiling are completely independent of the outer room and separated from it by a 6 in. air space. The inner walls have acoustic tiles. It has double doors, and the floor is heavily carpeted. The control room has only one brick wall with acoustic tiles covering it. It has a single solid door, "sealed" at the top, sides, and bottom. In the control room we have noiseless fans and in front of the doors of the examining room an air-conditioning unit; thus when the doors are open the air goes into the room and its temperature is kept cool and comfortable, which is very important in our climate.

Our testing equipment in the examining room is a standard audiometer, a microphone, two headphones, and two loud speakers. In the control room we have a standard pure-tone audiometer, a standard speech audiometer, a headphone, and amplifier, which are connected to the microphone of the examining room, having thus a talk-back system. The speech audiometer may be connected to either the headphones or the loud speakers of the examining room. We can deliver through the speech audiometer either live voice or records and through the standard audiometer pure tones and masking noise.

The pure-tone audiometry is tested in the examining room. Its value depends to a large

extent on the proper calibration of the audiometer and on the use of a good testing technique. If it is found by repeated audiograms on normal hearing persons that threshold readings are far from the normal (20 db. or more), the audiometer should be sent to the manufacturer for recalibration. We are fortunate to have in Havana an engineer, Mr. J. M. Manzini, who takes care of our calibration. If our threshold readings seem to be accurate "physically," from the engineer's point of view, the audiometer should be calibrated "clinically" in our testing room: first, the air-conduction system, simply by testing 10 or 15 young adults with normal hearing, finding the 10 or 15 average readings for each frequency, and thus setting our normal clinical threshold.

In a pure perceptive loss, the impairment by air conduction and by bone conduction should be equal. Using this fact as a basis, in order to calibrate the bone system, as suggested by Dr. Raymond Carhart, we take 10 or 15 mild or moderate cases of bilaterally symmetrical nerve loss (some presbycusis cases are good), test them first by air and later by bone without masking, following the principle of no lateralization of sound, no masking. The averages of the air and bone curves for each frequency are plotted on an audiogram, and the differences between those averages for each frequency are computed, and, finally, on another audiogram the corrected bone-conduction curve is plotted. That correction must be applied when patients are tested with the bone system. The reason for taking moderate cases of nerve losses is in order to avoid the background noise. With the air and bone systems "clinically" corrected, the threshold curves must be modified accordingly and the allowances found should be transported to the audiogram sheet to be kept. It is convenient to paste on the audiometer the number to add or subtract to each frequency. Daily check of the calibration of the instrument is a necessity. It is a good practice to have the technician check the audiometer on herself, both by air and by bone, before examining patients.

A good testing technique is also important. The tester should thoroughly familiarize herself with her audiometer. The air curve is found first by testing from 125 to 8000 cycles. In the first audiogram we explore up to 12,000 cycles to determine the upper limit. For practical purposes it would suffice to explore from 300 to 3500 cycles, the ones found on the telephone. The bone conduction is explored between 250 and 4000 cycles. The limits of the audiometer should be known. In most of them the low- and high-pitch tones by air do not go beyond 65 to 70 db., and by bone the intensity ranges from about 40 db. at 250 to 60 db. at 4000.

It is a good practice to start testing at 1000 cycles and to test the higher tones first (2000, 3000, 4000, 6000, 8000, and 12,000) because usually they are heard better, and next the lower tones (500, 250, and 125). The ascending-threshold method is the one we prefer, going from silence to hearing. But first the tone to be tested should be given to the patient at an intensity higher than his probable threshold, to be sure he hears it and thus knows what to expect. We call this identification of the tone. Afterward the tone should be dropped down quite abruptly (far below threshold) and then increased, ascending gradually, using the interrupter a great deal, to be sure the patient is answering right. The interrupter should always be used in changing frequencies as well as in changing intensities. We do not use the patient's signal, but have him raise his right or left index finger, depending on the ear being tested. The patient should be placed in such a way so he cannot see the control panel of the audiometer, but the examiner must be seated so as to be able to watch the patient's face and thus obtain additional clues. The examiner must be careful that the patient does not obtain any clues from him.

Enough time should be allowed for a pure-tone audiogram if one expects accurate results. If a spot check seems to be needed, only test by air the frequencies 500 and 4000 in both ears. If these are heard normally, it usually means that the patient has normal hearing. If the patient has tinnitus, the puls-

ing technique can be used: present a tone for just a few seconds and then shut it off. Repeat that three times and by then usually the patient is able to answer correctly. It is important to match his tinnitus as nearly as possible to some tested frequency and intensity. By doing that, one can check on future audiograms the status of the tinnitus to a certain extent. Tinnitus probably does not change the audiogram too much, and at any rate we obtain the threshold of hearing that the patient really has.

The bone conduction is tested exactly as the air conduction. We prefer a small hearing aid type bone conduction receiver, which is placed on the optimum site on the mastoid bone, found by actually trying it on several locations. Experience has shown us that in many cases the optimum place appears to be over the mastoid antrum, behind the upper portion of the auricle, although not in all cases.

Masking is done to incapacitate temporarily one ear, because if no masking is used a certain amount of sound might be transmitted to the ear not tested. But if too much masking is used, the threshold will be modified, because when an ear is blocked, it becomes very sensitive. We mask when there is a great difference between the two ears (35 to 50 db.), when both right and left curves look very much alike (shadow curves), and when the sound lateralizes to the ear not being tested. This applies to air conduction as well as to bone conduction. If we use masking because we have to, routinely we do tests with and without masking. At times, even when masking, the sound lateralizes to the ear masked. It should be found how much masking was used and write it down on the audiogram sheet. Masking should always be applied by air, and a simple rule is to place the masking sound in the poorer ear, increase its intensity until heard, and then raise it 10 to 15 db. more. Place it on the better ear, and one is ready to test the poorer ear.

Theoretically the best masking sound is the white noise, as it has all the frequencies, but actually it does not give enough masking, producing a threshold shift. Saw-tooth noise is the one used in most commercially available audiometers, but usually it is not rated adequately, and its intensity goes up to 50 or 55 db., but it does not remain the same for all frequencies. So it should be kept in mind that many times it is not possible to mask completely the ear not tested.

In the air curve we find the pure tone average (P. T. A.) and the best binaural average (B. B. A.). The P. T. A. is the average loss for the speech frequencies (500, 1000, and 2000). It is found in each ear separately by adding the loss of those three frequencies and dividing the sum by three. This is the procedure to follow when the air curve is more or less horizontal. But if the audiogram is a high- or low-tone loss basically, one should add the two highest points (least loss) of the speech frequencies and divide the sum by two. This also applies to any air curve which looks more or less peculiar. If one takes the highest points (least loss) of both air curves (right and left) of the speech frequencies, adds them up, and divides the sum by three, the best binaural average is obtained. Later we will see the importance of the P. T. A. and of the B. B. A.

In general it can be stated that the air curve in the audiogram incorporates all the factors of the hearing of a patient, while the bone curve gives a picture of the cochlear function, of the nerve function. Both curves can be obtained in a very accurate manner, but, as with any other test, one must be aware of the fact that there is a margin of error present in pure-tone audiometry.

In order to evaluate the hearing of a patient it is necessary to determine both air and bone curves, because the air curve by itself does not say too much. The relative positions of the air and bone curves are very important. Bone normal or increased and air depressed means a pure conductive loss. In conductive losses there is an air-bone gap, and this gap constitutes the conductive component. The maximum possible air-bone gap in a pure conductive loss is from 50 to 60 db. If there is decreased bone conduction and air

conduction, the loss is a result of an inner-ear lesion. The perceptive component will be the gap between the normal bone threshold and the bone curve obtained. In pure perceptive losses the loss by air is about the same as the loss by bone, thus both curves interweave or superimpose on the audiogram. But in conductive lesions the bone curve does not give a complete picture of the cochlear function, as fine details are not given because of the mass, stiffness, and friction factors present. The true picture can be arrived at if we subtract the mechanical factors present in conductive lesions. It was found that bone-conduction acuity improved after fenestration surgery because the mechanical factors were changed by the surgical procedure. The lever mechanism that the normal ear has to convert waves of a large amplitude and small force into waves of small amplitude and large force, known as the impedance-matching mechanism, was removed partially, the mechanical factors were thus changed, and there was a loss of 20 to 30 db. It was also found that stapes fixation seemed to produce a notching in the bone curve because of the mechanical factors, the well-known Carhart notch. Therefore, in order to have a true picture of the cochlear reserve, we must add to the bone curve the loss which was found to be due to mechanical factors, and thus one should add to the bone curve in decibels 0 to frequency 250, 5 to 500, 10 to 1000, 15 to 2000, and 5 to 4000. This is true only in cases of otosclerosis. Other conductive lesions show also mechanical factors, for example, adhesive otitis, but they are not well known yet and can not be treated audiometrically as otosclerosis. Better bone than normal in low frequencies and a little below in the high frequencies is quite typical of middle-ear disease, as happens in chronic suppurative otitis media.

The air-curve configuration will always tend to satisfy the impedance formula. So far as the auditory system is concerned, impedance is an interaction between stiffness and mass for any given frequency, as friction plays no part. Soon, perhaps, the classically known conductive deafness will be more properly called impedance deafness. The normal air curve will be horizontal, in the zero line. If there is an increase in stiffness, the impairment will be more pronounced for the low frequencies than for the high, thus having a low-tone air curve. If there is an increase in the mass factor, the impairment will be more pronounced for the high frequencies, thus it will be a high tone loss air curve. For example, in otosclerosis, when stapes fixation begins there is an increased stiffness (movement of the footplate of the stapes "stiff"), giving a low tone loss air curve. As the disease progresses the air curve first gets to be horizontal (below normal), and when fixation is completed there is an increase in the mass (movement fixed completely) and there is a high tone loss curve.

In acute tubal obstruction, such as in aero-otitis, the stiffness factor will give a low tone air curve. In secretory otitis, the mass factor increases, and gives a horizontal below normal curve first, and later a high tone loss air curve. In general any pathological condition in which fluid or pus covers the round window will give an increase in mass and subsequently a high tone loss curve.

If there is a tympanic perforation, the impedance-matching mechanism, which causes a marked differential in sound pressure at the two windows, resulting in good mobility of the intralabyrinthine fluids, will be affected and there will be a marked conductive loss, because sound will enter both windows with less of a differential in sound pressure. If sound would enter and hit both windows at exactly the same time, there would be no hearing because there would be no mobility of the inner-ear fluids.

We wish to insist that the air curve by itself is of a very limited value. For example, an air curve with a high-tone loss might be a suppurative otitis media, secretory otitis, otosclerosis, external meatus block, nerve deafness, and conduction deafness with associated nerve involvement.

In labyrinthine hydrops in the early stages, there is found a typical low-tone loss

for both air and bone. The typical audiogram of acoustic trauma is a notch at 4000, which deepens and widens as the process continues.

In the state of our present knowledge we think that an audiogram should not be interpreted just by its air and bone configuration, but by adding up the history, physical examination, laboratory tests, and the rest of the hearing tests. It is a good practice to try to find with the pure-tone audiometer and next with the speech audiometer the comfortably loud area, which normally is about 45 db. above threshold (comfortable loudness), and the uncomfortable loudness area, which normally is about 70 db. above threshold. In many cases we cannot do this with the standard commercially available audiometers because their intensities do not go high enough, but in some hearing losses that relationship is changed and loudness increases much more rapidly than normal as the intensity of sound is increased above threshold. This phenomenon is the well-known recruitment. In some of those cases we can find the comfortable and uncomfortable loudness areas very close, which is of importance, particularly for rehabilitation purposes.

In regards to testing recruitment, our opinion is that only the direct methods are practical and thus available to the practicing otolaryngologist: Fowler's alternate binaural test and Reger's equal loudness contour test. All the indirect methods, such as the difference limen tests, are excellent in theory because many deaf persons have neither one normal ear nor one unimpaired frequency, which are musts for the direct methods, but in practice all the indirect measures are complicated, requiring special equipment and personnel, and, what is more important still, the indirect methods are changing concepts continuously in the process of improvement and maturation. Perhaps soon a practical test for recruitment will be designed which will be applicable to all cases.

To use the alternate binaural test, there should be a difference of at least 20 db. and preferably 30 to 40 db. between the two ears. A tone close to the better ear threshold is presented first, and next it is given to the impaired ear. With the intensity of the tone presented to the better ear fixed, the intensity of the impaired ear is increased or decreased until the patient reports that the tone is equally loud in both ears. The same procedure is repeated at several levels above the threshold of the better ear. Results can be transported to the audiogram directly by drawing lines between points equally loud: the loudness-balance lines. If the lines remain parallel, there is no recruitment. If they converge, there is. The best way to do this test is with two closely matched audiometers or a twin circuit audiometer, but it can be performed with a single audiometer, although it is more difficult. Reger's equal loudness contour test needs only one frequency to be normal in the ear to be tested. Two tones of different frequencies are alternately presented to the same ear: one tone at the normal frequency, the other at any impaired frequency. The intensity of the tone at the normal frequency is fixed, and the intensity of the tone at the impaired frequency is varied until the patient reports that the two tones are equally loud. The same procedure is repeated at several levels, and by comparing the normal frequency with every other frequency, an equal loudness contour can be drawn at each level. If the contours remain parallel, no recruitment is present, but if they converge, there is recruitment.

Recruitment is confined to lesions of the organ of Corti. It is thought to be present in acoustic trauma, labyrinthine hydrops, congenital hereditary nerve deafness, and secondary nerve degeneration in otosclerosis and in otitis media. It is thought to be absent in acoustic tumors, lesions in the cerebellopontine angle, presbycusis, streptomycin loss, psychogenic loss, and pure conductive losses.

SPEECH AUDIOMETRY

As persons usually do not hear pure tones in their everyday lives, but mainly speech, it is necessary to find out how much hearing

there is for speech and how much understanding there is of the material heard—that is, to know how well a person is getting along socially from the hearing point of view. For that purpose, speech audiometry was designed.

The purpose of the speech reception test (S. R.) is to test the acuity or loss of hearing for speech. The test material is easy to understand, dissyllabic words, all of very nearly the same difficulty and therefore particularly suited to measure the threshold for speech. The Harvard Psycho-Acoustic Laboratory selected 84 words (two groups of 42 each) which are highly homogeneous, and are known as "spondees," which means that the two syllables of each word are equally stressed. Perhaps the best known phonographic version of the spondee words are Auditory Test Nos. 9 and 14 from Harvard and the lists W-1 and W-2 recorded at the Central Institute for the Deaf at St. Louis. Many lists of sentences, as well as lists of words, have been compiled for this test in the English language. We are working with a list of Spanish words somewhat similar to the Auditory Test No. 14, which basically is a modification of Dr. J. M. Tato's lists. We are in the process of calibrating our words with our equipment in our examing room with normal and hard of hearing persons, and thus far have established values which for the time being are only useful to us, as the whole procedure needs to become standardized.

The words can be administered by the use of live voice through a receiver, to which is attached a sound-level meter. By working the dial of the meter, the examiner knows whether or not he is delivering them correctly. This is known as monitored live voice. It is possible to train an intelligent girl to do properly this type of work as well as the pure-tone audiometry. In the United States, it is a relatively easy procedure because there are excellent hearing clinics which as part of their work do train personnel. I have visited one of the best, the Northwestern University Hearing and Speech Clinic, and I believe that they offer a basic certificate and an advanced certificate in hearing.

The other method to deliver words is by "recorded speech" using records or wire or tape. We prefer the live-voice method because it is faster, it has a greater flexibility, the tester can go over the words again if necessary, and one can check and recheck. Of course, two rooms are needed but this is necessary if the practicing otolaryngologist is to do hearing-aid evaluations, test children with auditory disorders, etc. With the use of records only one room is necessary; they are easier to administer because a person with special training is not quite so necessary, but, on the other hand, in our experience records do not last very long. We began using records in Spanish several years ago. They were recorded at the Northwestern University Hearing Clinic for experimental purposes, but lately we only use the live-voice method, which, in our opinion, is much better clinically. Perhaps a tape recorder will give better service than records.

The records may have all the words recorded at the same level, i. e., the constant level records, such as Auditory Test No. 14 and W-1. Other records give several words (three to six) at one intensity, and then the intensity decreases (1 to 4 db.), such as in Auditory Test No. 9 and the new W-2, which are of this descending type. When we used records we preferred the constant level type, thus being able to control the intensity ourselves. Those lists, either by the live-voice method or by recorded speech, can be delivered to separate ears (right ear with left ear covered with a dummy phone, and vice versa) or to both ears at the same time through the loud speakers (free-field).

The technique we use is as follows: The tester is in the control room and administers the words through the microphone of the speech audiometer, watching the dial of the VU meter. The output of the speech audiometer is connected to the headphone being worn by the patient, who is seated in the examining room. The patient repeats the words heard through his microphone, part of the talk-back system, while the tester

hears them through the earphone she wears in the control room.

We usually do the pure-tone audiometry first, thus we know the pure-tone average (P. T. A.). Through the speech audiometer, the tester starts to give the words 25 to 30 db. above the pure-tone average (25 db. if live voice is used; 30 db. for records). By doing the above, one is sure that the patient hears well the words given. Gradually the intensity is lowered by moving the decibel dial of the speech audiometer, and the threshold for speech will be the point at which a patient repeats correctly 50% of the material given. A practical method is having the word lists in front of the tester, in groups of five words; the speech threshold (S. R.) will be arrived at when the patient repeats correctly 5 out of 10 delivered words. The S. R. test determines the lowest level at which a patient is able to hear speech of common usage. The relation between the S. R. and the P. T. A. should be approximate in most patients using both English and Spanish words. If Spanish words are used, it should be within 10 db. This consistency is found in conductive as well as in perceptive losses. Thus the S. R. test checks our air-conduction audiogram. If the S. R. test is better than the P. T. A., it might mean psychogenic deafness, apuretonia, or malingering and has been found in some cases of head injuries. On the other hand, the P. T. A. might be better than the S. R., and this is seen in cases of language difficulties or educational or intellectual deficits. The speech reception threshold is a good indication of the individual ability to follow up everyday connected speech. The S. R. threshold is more reliable than the discrimination thresholds. It is very important to use standardized presentation, control level, and test material. If records are used, first calibrate the speech audiometer with the 1000-cycle leading tone. If live voice is used, first use the record just to calibrate the system. Our normals, in our set-up, with our equipment, and with our words, run so far approximately as follows:

Normal: 0 to 15 db.

Mild hearing loss: 15 to 30 db.

Moderate hearing loss: 30 to 60 db.

Severe hearing loss: 60 to 80 db.

Very severe hearing loss: more than 80 db. (not much to do in the way of amplification)

After the determination of the S. R. threshold, the tester determines the ability of the patient to make fine discriminations. This is done by the phonetically balanced test (PB test), which is not a threshold, but a test of ability to understand, or intelligibility. The test material consists of the PB-50 word lists, devised at the Psycho-Acoustic Laboratory of Harvard. There are 20 lists, and each list has 50 monosyllabic words difficult to understand, all of which must be delivered. Each list is phonetically balanced, which means that the list as a whole reproduces the main phonetic types encountered in spoken English. The most popular phonographic version has been the Rush Hughes version. The W-22, with 12 lists, was recorded at the Central Institute for the Deaf.

Again, we are working in Spanish with a list somewhat similar and are trying to standardize it in our room with our equipment and with normal and hard of hearing persons.

The technique is as follows: A carrier phrase should be used before the word, such as "you will say," and the 50 words are fed to the patient at 25 db. above his speech threshold, because one has to be sure he hears the words. If records are used, the intensity should be 30 db. above the S. R. threshold. The result is the number of words missed, and it is given in percentages. A practical way to do it is by giving the patient 2% for each word repeated correctly. The total score is known as the PB Max.

In pure conductive losses, such as in otosclerosis without nerve degeneration, the PB score is excellent. In most perceptive losses there is some reduction in the score. In labyrinthine hydrops there is very poor discrimination; the PB score is poor. In high-

tone deafness one should expect poor discrimination, particularly if the 2000 to 3000 frequencies are involved. Presbycusis frequently shows poor discrimination, and in some cases there is phonemic regression: a person with a loss of the perceptive type and speech threshold agreeing with the pure-tone average but lower scores in discrimination than expected, without having the intellectual capacities deteriorated. Gaeth coined the term and studied the problem. Our discrimination scores are running around these figures:

Normal: 92%-100%

Mild loss: 85%-92%

Moderate loss: 75%-85%

Severe loss: 50%-75%

Very severe loss: less than 50% (less than 30%, not much to do in the way of amplification with a hearing aid)

HEARING-AID EVALUATION

The rehabilitation of the deafened and hard of hearing adult can be successfully carried out in the office of the practicing otolaryngologist. The obtaining of important clues to compensate the hearing loss by attentively observing the speaker's lips (lipreading or speechreading), of obtaining the maximum benefit of the hearing present by auditory training, and the preservation of the speech present by speech training undoubtedly require a highly trained personnel, who can be obtained if the otolaryngologist is specially interested. However, the same technician who does the pure-tone and speech audiometry can also do the hearing-aid evaluation. This is of upmost importance, because the most useful tool for rehabilitation purposes that we can offer those patients to whom no medical or surgical treatment is indicated is the hearing aid.

It is best first to explain to the patient in detail how the aid works. If the aid to be tested is not an all-transistor type, the batteries are tested with a hearing aid battery tester, available commercially for a few dollars, and good to test A and B batteries. The ear mold is inserted in the ear to be tested. The mold is a standard full mold with standard bushings available commercially in five sizes for each ear. The molds are well kept in green soap overnight. It is a good practice to have the patient open and close his mouth several times, to be sure the ear mold fits him properly. The air conduction is the fitting of choice for most patients, with the exception of those having severe external otitis or discharge, in whom an earpiece inserted in the auditory canal would be a contraindication.

The patient is seated in the examining room. The aid to be tested is situated on an acoustic tile, and it is kept in place with the aid of rubber bands on back of the patient and about 58 in. higher than the patient's head. The loud speaker is in front, at about 68 in. from the chair and at the same level as the patient approximately. After the patient is seated with the aid on, the examiner talks to him and explains how to graduate the intensity control of the aid until it sounds comfortably loud. When the patient is satisfied with the adjustment, the tester goes to the control room and gives by free field the speech-reception list and the discrimination list which were given previously, in order to compare both results of the S. R. threshold and the PB Max with and without the aid.

Next another PB list is given, but this time with some masking noise in the background at an intensity of about 15 db. above the S. R. threshold. In this way we test the performance of the aid in conditions that are far from ideal. The pain threshold is explored by talking to the patient and explaining that we are to talk louder and louder (and actually doing it) until the patient states that he is having discomfort and pain. Both levels are noted to determine the tolerance limit and make pertinent recommendations.

In this fashion several aids are tested, but if the otolaryngologist has difficulties in obtaining aids in his community, he can get a master hearing aid, such as the one described by F. Guilford, of Galveston, Texas, and which can be procured commercially.

In general it can be said that the best way to know how an aid will work is by actually

testing it, because, in our experience, sometimes the hearing aid does astonishingly well in cases in which we did not expect such a good performance. The best possible case is a pure conductive loss (35 to 55 db. loss) with good nerve and good discrimination. In mild loss cases (close to 30 db. loss) it is necessary to explore occupational needs, such as in the case of a librarian or a priest, who may profit with an aid even if their loss is as low as 15 db. They may become part-time hearing-aid users. If both ears show a loss between 30 and 65 db., the aid should be worn in the worse ear so that the better ear can be of some use. If there is a big difference between the two ears, the aid will not work properly, such as, for example, a 25 db. loss in the better ear and an 80 db. loss in the poorer. But it will work if the difference is not too great, such as a 25 db. loss in the better ear and about a 40 db. loss in the poorer.

In cases of severe unilateral deafness, most adults will have problems of localization, particularly if the onset of the loss was sudden. Children with equal losses seem to localize much better. The advice to the adult should be to learn to maneuver the better ear, placing it on the best spots always after panning quickly to see where the sounds come off. In noisy places sometimes it is better to place the better ear close to a wall so it may act as a reflecting surface for normal conversation. Lipreading and auditory training should also be advised.

If one ear or both have a loss greater than 65 db., the better ear is the choice for an aid. However, an aid can rehabilitate up to 65 db. approximately. If the loss is much greater, such as 80 db., no hearing aid will take the patient to the range of normal hearing. A carefully selected powerful instrument should be prescribed, and lipreading and auditory training are musts. Thus far the aids with tubes seem to work better for severe losses than the new transistors.

If the P. T. A. is about the same in both ears and also the S. R. and the PB Max, the right ear is preferred, providing the patient uses the telephone on his left ear. The speech frequency is an important item to consider to determine how the aid will work. If, for example, the frequency 500 is normal and an abrupt drop comes in the 1000 frequency, the aid will not work well because it cannot be so selective as to amplify low or high tones, and lipreading should be recommended. If there is a deep notch at 4000, most probably the patient will not be bothered much, having only certain difficulties, such as not hearing well the telephone ring, which can be changed into a buzzer type.

Even in those patients with a much better bone than air conduction, the air-conduction type of aid works better in about 95% of the cases. If the loss by bone is 15 db. or more, 100% of the cases will benefit more with the air-conduction type. Some otolaryngologists will make their own molds, others will let a reputable dealer do it. At any rate, one should be careful that it is properly fitted, not too loose, because the low frequencies will be lowered more and more as the ear mold becomes looser, and it will squeal. When sound escapes, it may reach the microphone and be picked up and amplified: the acoustic feedback. On the other hand, if the ear mold is too tight, it will cause great discomfort, particularly with the movements of the jaw. After the prescription of the aid is made, the patient should be sent to a reputable dealer. He can be relied upon to recommend the particular combinations of amplifier, earphone, and tone control setting that seem to work best for the patient. It is important to regulate the maximun output to avoid tolerance problems. The otolaryngologist should work as close as possible with the reputable dealers.

After the aid is purchased the patient has to be well trained in the use of his aid, and stress should be made that the aid has to be used gradually, first in the patient's room identifying noises, later with the family and friends, and, finally, in difficult situations, such as in a movie.

The deaf should be viewed as a total problem. If nothing can be done by medical or surgical means, one should insist on a hearing aid, if at all possible. In cases with poor

discrimination both lipreading and a hearing aid should be advised, although those patients will have some difficulties, such as distortion. Apparently things occur somewhat like this: If a patient uses only lipreading, he understands 20%, with a hearing aid alone 40%, and, using both, he will understand not 60% but at times as much as 85%. It is very important to correct, if necessary, the psychological problems present in many cases, which may develop into serious personality changes if not taken care of or referred to the proper specialist.

VESTIBULAR TESTS

The extent of the vestibular examination will depend mainly on the patient's symptomatology and on the findings of the hearing tests. As a routine we prefer the caloric test because each labyrinth can be tested separately. We use Kobrak's test of minimal caloric estimulation as modified by W. J. McNally. If there is a perforation present, we prefer the Dundas Grant cold-air douche. Both tests are very well described in the manual published by the American Academy of Ophthalmology and Otolaryngology titled "Examination of the Labyrinth in Relation to Its Physiology and Nonsuppurative Diseases," by W. J. McNally and E. A. Stuart, 1953 edition. However, if an intracranial lesion is suspected, the mass caloric irrigation is preferred, because it is felt that other tests used to study the function of the vestibular apparatus are not sufficient for the purpose of determining the presence of intracranial lesions. The technique used is the one advised by Benjamin Shuster, and it is described in detail in the ARCHIVES OF OTOLARYNGOLOGY (**51**:831-851 [June] 1950) by Fernando G. Santamarina.

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Adrenosem in the Control of Hemorrhage from the Nose and Throat

A Preliminary Report

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The control of spontaneous and postoperative hemorrhage from the nose and throat is a matter of concern to the otolaryngologist and the patient alike. In some instances the control of bleeding from these areas may be a relatively simple matter limited to some topical therapeutic procedure. On other occasions the patient's course may dictate more radical and even life-saving measures. In either event, any therapeutic agent or procedure which facilitates control of hemorrhage is a welcome addition to the otolaryngologist's therapeutic armamentarium.

In past years efforts have been concentrated primarily on the production of substances calculated to act on the blood-clotting mechanism, either by systemic administration or by local application. Such of these agents as I have employed have afforded rather poor results for the most part, and recourse to more time-honored, but troublesome, methods has been the usual rule. There now appears on the horizon a new product which gives promise of being a valuable drug in the control of bleeding from the nose and throat. This product is Adrenosem, and it exerts its action on the capillary wall, rather than through the blood-clotting mechanism.[1] This paper purports to set forth my experience with the use of Adrenosem in the control of postoperative hemorrhage and in the control of selected cases of epistaxis. In none of these cases was there any reason to suspect an abnormal clotting mechanism of the blood.

Adrenosem is a synthetic chemical with the full chemical name of adrenochrome monosemicarbazone sodium salicylate complex. Adrenochrome is an oxidation product of epinephrine and is very unstable. A derivative of adrenochrome, the monosemicarbazone, is a very stable product in dry form or in aqueous solution, but it is only slightly soluble in water. A stable aqueous solution of adrenochrome monosemicarbazone, suitable for oral or parenteral administration, may be prepared by using sodium salicylate as a solubilizing agent. The complex is referred to as Adrenosem.

From information supplied by the manufacturers * and from the publication of Sherber [1] the following pertinent data are recorded for Adrenosem regarding its chemical relationship, pharmacology, and the clinical indications and contraindications for its use.

CHEMICAL RELATIONSHIP

Adrenochrome monosemicarbazone, being an oxidation product of epinephrine, is a distant chemical relative. One can assume that it would also be associated very closely with the adrenal medulla. Since it has been postulated that corticotropin (ACTH) and cortisone stimulate only the adrenal cortex and have no effect on the adrenal medulla, there is no reason to believe that either corticotropin or cortisone would have any marked hemostatic effect. Conversely, there is no evidence that Adrenosem will stimulate or alter in any way the activity of either the pituitary or adrenal cortex.

* The S. E. Massengill Company, Bristol, Tenn.

PHARMACOLOGY

Although chemically related to epinephrine, Adrenosem has no sympatheticomimetic action. It will not significantly raise blood pressure or affect the cardiac rate or volume. Being a systemic hemostat, Adrenosem has no effect on blood chemistry and in no way interferes with the blood-clotting mechanism or its components.

Concomitant administration of vitamin K will not interfere with the action of Adrenosem, and vice versa. Paradoxically, Adrenosem can be administered with heparin or other anticoagulants, neither interfering with the action of the other.

Adrenosem is prepared for oral or systemic administration. Because of rapid destruction and elimination in the gastrointestinal tract, the oral dose is approximately one-third as effective as the parenteral dose.

Because it is a hypertonic solution, Adrenosem should not be used intravenously. An intramuscular injection is absorbed very rapidly and is accompanied by a painful stinging sensation of brief duration.

There are no cumulative effects from the continued and prolonged use of Adrenosem, although a residual effect on capillary permeability persists for a limited time. Adrenosem is rapidly oxidized in the body and eliminated completely wthin a 12-hour period. The hemostatic effects may be noted for a 48-hour period after complete elimination. This is borne out in clinical cases of hereditary telangiectasia, in which, after injection, bleeding is stopped in 48 hours but starts again in 72 hours if no intervening doses of Adrenosem are given. In hereditary anomalies, such as telangiectasia, Adrenosem does not have a curative action, but produces a temporary reversal of symptoms.

Extensive studies of the parent compound, adrenochrome, and related compounds show that the monosemicarbazone is excreted to the extent of 50%-70% through the kidneys. About 20%-30% is excreted as an extra indole. Certain strains of intestinal bacteria, especially Escherichia freundii, which may also be present in the urine, destroy adrenochrome semicarbazone, with the formation of indole. In subjects in which the intestinal tract is freed of bacteria by administration of sulfaguanidine, little or none of the adrenochrome derivatives are converted into indole; hence the formation of indole is due to bacterial action. In humans 8%-28% of the semicarbazone is excreted in the urine in five to six hours.

Attempts have been made to check bleeding with Adrenosem in pathologies characterized by blood dyscrasias. Adrenosem is only partly successful in these cases. Authorities agree that in all types of purpura there exists a capillary liability in that excessive permeability is always present. In addition to this, in certain types of purpura, the blood-clotting mechanism may also be at fault. This is notably true in thrombocytopenia and pseudohemophilia. Adrenosem does not correct the faulty clotting mechanism but does correct the excessive permeability of the capillaries. Therefore, in certain types of purpura, Adrenosem can be regarded as a treatment for one of the symptoms present.

In hemophilia, in which it is believed that the bleeding tendency is due solely to a blood anomaly and that the capillaries are normal, Adrenosem is of no significant value.

It is known that sodium salicylate, if taken in large enough doses, will interfere with the blood-clotting mechanism. Although sodium salicylate is present in Adrenosem, the amount is so small that the physiological action is insignificant.

Adrenosem is therefore specific for conditions characterized by capillary permeability. It checks bleeding from a broad capillary bed by causing a correction of excessive permeability and an increase in capillary resistance. In clinical studies it was noted that Adrenosem promoted the retraction of severed capillary ends, thus aiding in retention of a clot, even though the clot is not well organized. The capillary walls cannot be made impermeable or excessively dense. They are simply returned to normal permeability. Adrenosem has no effect on large severed blood vessels and arterioles. Since

it is not a vasoconstrictor, it has no effect on the muscle sheath of arteries and arterioles; therefore arterial bleeding is not controlled by Adrenosem.

CLINICAL INDICATIONS

The administration of Adrenosem has been found to be of value in the control of bleeding in such conditions as idiopathic purpura, retinal hemorrhage, familial telangiectasia, epistaxis, pulmonary bleeding, petechial hemorrhages incident to x-ray therapy, metrorrhagia, and menorrhagia. It does not affect the underlying pathologies.

CONTRAINDICATIONS

Adrenosem has a very high index of therapeutic safety; at recommended dosage levels there are no contraindications. No evidence of toxicity has been found in laboratory animals. It has been determined that the L. $D._{50}$ in experimental animals is 183 mg. per kilogram of body weight.

Adrenosem has no cumulative effects. Patients treated for periods of over two years showed no toxic effects attributable to the drug. Volunteer subjects have received injections of 25 mg. at one dose and showed no untoward reactions.

There is evidence to show that the effects of Adrenosem are inhibited by the action of the antihistamines. For Adrenosem to be effective, the patient should have no antihistamines for 48 hours before administration.

My first experience with the use of Adrenosem occurred on Nov. 20, 1953, when it was employed to control a case of primary postoperative hemorrhage from the adenoid region of a child. The results were so dramatic that since that date Adrenosem has been used preoperatively to reduce bleeding from all otolaryngologic and bronchoesophagologic procedures, to treat postoperative hemorrhage from the tonsil and adenoid regions, and to treat selected cases of epistaxis. The number and variety of conditions are recorded in Table 1.

In order to more carefully evaluate the effectiveness of Adrenosem, the available records of all tonsil and adenoid operations performed during 1952 and from January through Nov. 19, 1953, before the use of Adrenosem was begun, were reviewed. Certain data have been tabulated as to the total number of primary and secondary tonsillectomies and adenoidectomies performed on both children and adults, average operating time, total number of patients requiring sutures, and the total number of cases of primary and secondary postoperative hemorrhage. Patients 16 years of age and over were classified as adults. Bleeding which occurred in the first 24 postoperative hours was classified as primary, and bleeding which occurred after this time was recorded as secondary in the conventional manner. This information has been recorded in Tables 2 and 4. A similar tabulation was made for all tonsillectomies and adenoidectomies performed from Nov. 20, 1953, through Aug. 31, 1954. These data have been recorded

TABLE 1.—*Number and Variety of Conditions Treated with Adrenosem from Nov. 20, 1953, Through Aug. 31, 1954*

1. Surgical Cases

Preoperative Treatment with Adrenosem	Cases, No.
Tonsillectomy and adenoidectomy	176
Submucous resection of nasal septum	8
Submucous ethmoidectomy	1
Excision of growth of nasal septum	1
Removal of foreign body from the external nose	1
Removal of growth from the nasopharynx	1
Removal of ranula from the floor of the mouth	2
Tracheotomy	2
Radical mastoidectomy	3
Removal of mixed tumors (parotid, cheek)	2
Diagnostic laryngoscopy	1
Laryngoscopy and removal of foreign body	1
Laryngoscopy and biopsy	3
Laryngoscopy and removal of benign growth	9
Diagnostic esophagoscopy	9
Esophagoscopy and removal of foreign body	13
Diagnostic bronchoscopy	4
Bronchoscopy and removal of foreign body	1
Nasal fractures	2
Caldwell-Luc operation	2
Active Treatment with Adrenosem	
Postoperative tonsil and adenoid bleeding	7

2. Medical Cases

Active Treatment with Adrenosem	Cases, No.
Epistaxis (choanal region of nose)	13
Traumatic hemorrhage from larynx	1
Bleeding from a tonsillar crypt	1

TABLE 2. — *Tonsillectomies and Adenoidectomies Performed from Jan. 1, 1952, Through Nov. 19, 1953, Without the Preoperative Use of Adrenosem*

Type of Operation	No. of Patients	Total Operating Time, Min.	Average Operating Time, Min.
Primary			
Children	240	4,516	18.81
Adults	57	1,419	24.89
Secondary			
Children	12	215	17.91
Adults	4	107	26.75
Total	313	6,257	Average 19.99
Combined *	10		
Time not recorded	13		
Records lost	5		
Grand Total	341		

* Time was not recorded for combined operations.
Total number of patients requiring suture of tonsillar fossae at operation for control of bleeding: 28.

in Tables 3 and 5. It would appear that the results tabulated in Tables 2 and 4 represent a sufficient number of cases to serve as a standard for comparison. The total number of operations performed after the routine use of Adrenosem was begun is less than the total number of operations performed in the preceding two years before Adrenosem was employed. However, it is believed that the information on Adrenosem-treated cases is sufficient to serve as a basis for a preliminary report.

The same technique for tonsillectomy and adenoidectomy was employed throughout the entire period under consideration. In the cases of primary operation the tonsils were removed first with a Daniels tonsillotome, and the remains of the lower tonsillar pole were removed by snare. The adenoids were next removed with a LaForce adenotome. The soft palate was then elevated, and the remaining lymphoid tissue in the fossae of Rosenmüller, vault of the nasopharynx, lips of the torii tubarii, lateral pharyngeal bands of lymphoid tissue, and lymphoid extensions on to the posterior surface of the soft palate, when present, were removed under direct vision with punch forceps. The nodules of lymphoid tissue on the posterior wall of the mesopharynx were likewise removed with punch forceps. The technique used is essentially that described by Reeves and Brill.[2] Dental rolls were inserted into each tonsillar fossa and into the nasopharynx after removal of the tonsils and adenoids, and when these were removed, these areas were swabbed with a 10% solution of tannic acid and glycerin, and the excess aspirated. The same technique was employed for secondary operations except that tonsillar tags were removed by snare.

The operative technique for any given operation listed in Table 1 remained the same throughout the entire period under consideration.

The type of anesthesia for tonsil and adenoid operations remained the same throughout the entire period preceding May 1, 1954. Children were inducted with vinyl ether (Vinethene) and drop ether, and anesthesia maintained by ether insufflation. All adults were given a general anesthetic, with induction by vinyl ether and drop ether or by cyclopropane, oxygen, and ether, after which anesthesia was maintained by ether insufflation. After May 1, 1954, all tonsillectomies were performed under endotracheal anesthesia. The results indicate that none of the factors mentioned in Tables 2, 3, 4, and 5 have been unduly influenced by the change in anesthetic technique. All mastoidectomies and removal of tumors were done with intravenous thiopental sodium (Pentothal So-

TABLE 3. — *Tonsillectomies and Adenoidectomies Performed from Nov. 20, 1953, Through Aug. 31, 1954, with Preoprative Use of Adrenosem*

Type of Operation	No. of Patients	Total Operating Time, Min.	Average Operating Time, Min.
Primary			
Children	148	2,508	16.94
Adults	20	412	20.60
Secondary			
Children	5	82	16.40
Adults	3	60	20.00
Total	176	3,062	Average 17.39
Combined	0		
Time not recorded	0		
Records lost	2		
Grand Total	178		

Total number of patients requiring suture of tonsillar fossae at operation for control of bleeding: 6.

dium) as the anesthetic agent, with the exception of one recent mastoidectomy done under endotracheal anesthesia. All nasal operations, and all laryngologic, bronchoscopic, and esophagoscopic procedures were done under local anesthesia.

Initially the plan of preoperative Adrenosem administration consisted of 1 cc. of solution injected into the gluteal region shortly after the anesthetic was started in the operating room or one hour before operation as the atropine sulfate. Older patients were given appropriate doses of morphine sulfate in addition, and those for surgery under local anesthesia were given appropriate doses of barbiturates. None of the drugs used concomitantly in the treatment of patients on Adrenosem therapy seemed to have any neutralizing effect on the Adrenosem.

In the treatment of medical cases the amount of Adrenosem given at each injection was 1 cc. containing 5 mg. None of the drugs

TABLE 4.—*Postoperative Tonsillar and Adenoid Hemorrhage Occurring from Jan. 1, 1952, Through Nov. 19, 1953, in Patients Without Preoperative Adrenosem*

Primary Hemorrhage		Secondary Hemorrhage	
Type	No.	Type	No.
Adenoid	7	Adenoid	4
Treatment:		Treatment: observation	
Postnasal pack	4		
Observation	2		
Clot removed	1		
Tonsil	1	Tonsil	0
Treatment: suture			
Undetermined source	1		
Treatment: spontaneous arrest			

TABLE 5.—*Postoperative Tonsillar and Adenoid Hemorrhage Occurring from Nov. 20, 1953, Through Aug. 31, 1954, in Patients with Preoperative Adrenosem*

Primary Hemorrhage		Secondary Hemorrhage	
Type	No.	Type	No.
Adenoid	2	Adenoid	3
Treatment: clot removed, Adrenosem		Treatment: clot removed, Adrenosem	
Tonsil	2	Tonsil	0
Treatment: clot removed, Adrenosem			

in the case of patients' being operated upon under local anesthesia. The latter plan was subsequently adopted for all types of operations. In the cases of submucous resection of the nasal septum, an additional injection was given on the patient's return to the room after operation and one hour before removing the nasal packing the following day. The brief discomfort which attends the injection of Adrenosem into the gluteal region has not been a significant problem in children or adults as originally anticipated. In about half of the patients the Adrenosem was given as a separate injection. In the remaining cases it was given in the same hypodermic used concomitantly in the treatment of medical cases on Adrenosem therapy seem to have any neutralizing effect on the Adrenosem. For the sake of completeness, these drugs have been listed in Table 6.

The following patients were treated in Lenoir Memorial Hospital or in the office if hospitalization was not necessary. In the case reports only the pertinent facts regarding history, results of general physical and local nose and throat examinations, and laboratory studies have been included. The blood serological test made on all older children and adults was negative in all instances.

REPORT OF CASES

SURGICAL CASES: POSTOPERATIVE TONSIL AND ADENOID HEMORRHAGE

Primary Adenoid Bleeding

CASE 1.—J. Y. Jr., a white boy, aged 6 years, had an uneventful tonsillectomy and adenoidectomy, which ended at 8:30 a. m. Nov. 20, 1953. He was returned to his room, where the oozing from the nose and mouth soon stopped. At 12:30 p. m. on the same day the patient sneezed and immediately began to bleed from the nose and mouth. He was taken to the operating room and one injection of Adrenosem given. After 20 minutes it was noted that the bleeding had almost stopped. The clots were then removed from the nose and nasopharynx, without anesthesia being given. A small clot re-formed in the nasopharynx, and when this was removed the field remained dry. Three additional injections of Adrenosem were given at two-hour intervals. The hemorrhage had stopped within 30 minutes after the initial injection of Adrenosem.

TABLE 6.—*Drugs Used Concomitantly with Adrenosem in the Treatment of Patients*

Case No.	Drugs Used with Adrenosem
1-7	None
8	Phenobarbital, mannitol hexanitrate (Nitranital)
9	None
10	Pentobarbital (Nembutal), phenobarbital, penicillin
11	None
12	Pentobarbital, phenobarbital, Pamine,* hydralazine (Apresoline), aluminum hydroxide gel (Amphojel), mannitol hexanitrate, ferrous sulfate
13	Phenobarbital, penicillin, Perihemin capsules,† Unicap Vitamins ‡
14	Secobarbital, meperidine (Demerol), penicillin, codeine
15	Phenobarbital, penicillin, belladonna tincture, magnesium magma (Milk of Magnesia)
16	Phenobarbital, pentobarbital, hydralazine
17	Phenobarbital, pentobarbital, penicillin, Agarol,§ cascara
18	None
19	Phenobarbital, pentobarbital, Mol-Iron,‖ codeine
20	Pentobarbital, phenobarbital
21	Penicillin, streptomycin
22	None
Unrecorded case	Vitamin K, ascorbic acid, rutin, ferrous sulfate, pentobarbital

* Epoxytropine tropate methylbromide.

† Each capsule contains ferrous sulfate exsiccated, folic acid, vitamin B_{12}, ascorbic acid, extract stomach powder, and extract insoluble liver fraction.

‡ Each capsule contains vitamin A, vitamin D, ascorbic acid, thiamine hydrochloride, riboflavin, pyridoxine hydrochloride, calcium pantothenate, and nicotinamide.

§ A fluid emulsion of mineral oil and an agar-gel.

‖ Each tablet contains molybdenum oxide and ferrous sulfate.

On admission to the hospital Nov. 19, 1953, the blood coagulation time was found to be 3 minutes and 25 seconds and the bleeding time 1 minute and 20 seconds.

CASE 2.—S. S., a white girl, aged 3 years, referred by Dr. J. Dawson, had an uneventful tonsillectomy and adenoidectomy, which ended at 9:45 a. m. Feb. 10, 1954. She was returned to her room, and the usual oozing soon stopped. At 12:30 p. m. the patient retched and began to bleed from the nose and mouth. The patient was examined at this time, but the bleeding had stopped spontaneously. At 1:45 p. m. she vomited about 6 oz. of old blood and began to bleed freely from the nose and throat. The patient was given one injection of Adrenosem and taken to the operating room. When it was noted that the bleeding had about stopped, the clots were removed from the nose and nasopharynx. The field remained dry. Adrenosem was then given at 30-minute intervals for three additional injections. The bleeding stopped within 30 minutes after the initial injection of Adrenosem.

On admission to the hospital Feb. 9, 1954, the blood coagulation time was found to be 3 minutes and 15 seconds and the bleeding time 1 minute and 30 seconds.

Secondary Adenoid Bleeding

CASE 3.—N. I. L., a white girl, aged 9 years, had an uneventful tonsillectomy and adenoidectomy performed Feb. 22, 1954. On Feb. 27, 1954, the patient picked the right side of her nose, and bleeding started but soon stopped to recur for three brief episodes during the day. The bleeding recurred on the morning of March 28, 1954. The patient was seen in the office at 3:15 p. m. March 28, 1954, and because of a mild excoriation of the right Kiesselbach's area, it was felt that this must have been the source of bleeding. The area was cauterized with acid. The bleeding recurred at 5:30 p. m., and the patient was hospitalized.

Examination of the patient in the room revealed a fresh blood clot in the nasopharynx, with active bleeding around the clot. One injection of Adrenosem was given, and when the bleeding was hardly noticeable, the clot was removed with sponge forceps. It re-formed slowly, but after the second removal there was no further bleeding. The time interval for control of bleeding was 30 minutes. Adrenosem was given every 30 minutes for two additional injections.

On Feb. 21, 1954, the blood coagulation time was found to be 3 minutes and the bleeding time 1 minute and 30 seconds. Laboratory studies made March 1, 1954, showed that the erythrocyte count was 3,720,000 and the value for hemoglobin was 9.5 gm. There were 9300 white blood cells, with 50% segmented cells, 42% lymphocytes, and 8% monocytes.

CASE 4.—G. T., a white boy, aged 17 years, referred by Dr. F. Norris, had an uneventful tonsillectomy and adenoidectomy June 21, 1954. The patient was discharged from the hospital June 22, 1954. He returned June 23, 1954, with a history of intermittent bleeding from the nose and mouth for about one hour.

Examination in the emergency room of the hospital revealed active bleeding from the nasopharynx. Two cubic centimeters of Adrenosem was given on arrival and the clot removed from the nose and nasopharynx three times over a period of 30 minutes. At the end of this time, the active bleeding stopped. Mild spotting from the nose continued about two hours after hospitalization. Adrenosem was continued at intervals of one hour for four additional injections. He was discharged from the hospital June 24, 1954.

The blood pressure on admission to the hospital was 110/70. The peak pressure recorded at any time was 125/80.

Laboratory studies made June 20, 1954, showed that the blood coagulation time was 4 minutes and 30 seconds and the bleeding time was 2 minutes. The erythrocyte count was 4,200,000, and the value for hemoglobin was 13 gm. There were 9600 white blood cells, with 76% polymorphonuclear forms, 20% lymphocytes, and 4% eosinophiles. A repeat on laboratory studies June 24, 1954, revealed an erythrocyte count of 3,740,000 and a hemoglobin value of 12.5 gm. There were 10,900 white blood cells, with 68% polymorphonuclear forms, 26% lymphocytes, 5% monocytes, and 1% eosinophiles.

CASE 5.—R. S., a white boy, aged 6 years, referred by Dr. Rachel Davis, had an uneventful tonsillectomy and adenoidectomy Aug. 18, 1954. On the morning of Aug. 26, 1954, he developed intermittent bleeding from the nose over a period of two hours.

Examination in the office revealed a small amount of fresh blood clot in the right nostril and nasopharynx, with slight oozing from the nose. The clots were removed, and the patient was hospitalized. He received one injection of Adrenosem at intervals of one hour for a total of three injections. The bleeding stopped within 15 minutes after the first injection of Adrenosem and did not recur. He was discharged from the hospital the following day.

Laboratory studies made Aug. 17, 1954, revealed that the blood coagulation time was 4 minutes and the bleeding time 2 minutes.

Primary Tonsillar Bleeding

CASE 6.—H. K., a white woman, aged 27 years, referred by Dr. S. Parker, had an uneventful tonsillectomy and adenoidectomy March 11, 1954. After return to her room, the usual oozing from the nose and mouth soon stopped. At 9:10 p. m. that night the patient vomited and began to spit up red blood.

Examination in the room a few minutes later revealed a large clot in the right tonsillar fossa, with bleeding around the clot. One injection of Adrenosem was given, and when the bleeding was seen to slow up, the clot was removed from the fossa with sponge forceps. It re-formed quickly, but after the clot was removed the second time the field remained dry. The time interval for cessation of bleeding was about 30 minutes. Adrenosem was given at 30-minute intervals for four additional injections.

On March 10, 1954, the blood coagulation time was found to be 3 minutes and the bleeding time 2 minutes and 15 seconds.

CASE 7.—A. D., a white child, aged 6 years, referred by Dr. J. Arnold, had an uneventful tonsillectomy and adenoidectomy, which terminated at 8:56 a. m. Aug. 13, 1954. The postoperative oozing on the patient's return to the room soon stopped. The course was uneventful until 8:30 p. m., when the patient vomited and began to bleed from the nose and mouth. One injection of Adrenosem was given at 8:45 p. m. and continued at intervals of one hour for a total of three injections. I examined the patient at 10 p. m., at which time all bleeding had stopped. A clot was removed from the right tonsillar fossa, and the further course was uneventful.

Laboratory study made Aug. 12, 1954, showed that the blood coagulation time was 4 minutes and the bleeding time was 2 minutes.

MEDICAL CASES

Epistaxis

CASE 8.—M. F. C., a white man, aged 55 years, referred by Dr. C. Wooten, was first seen in the office Dec. 15, 1951, bleeding profusely from the back of the right side of the nose. The bleeding was beyond control in the office, and the patient was hospitalized for insertion of a postnasal pack. This was removed five days later, and the patient discharged from the hospital two days later. The referring physician reported that the average blood pressure for several years had been 250/140.

The patient was seen a second time at 7 a. m. Dec. 15, 1953, in the accident dispensary of the hospital. He stated that he had been bleeding from the right nostril at intervals since midnight. A few minutes before his coming to the hospital the bleeding became profuse and constant. Examination revealed free bleeding from the right choanal region.

The patient was given Adrenosem at one-hour intervals for three injections, with considerable diminution in the bleeding. He was then hospitalized and the Adrenosem continued at hourly inter-

vals until 10 p. m. Dec. 15, 1953, then at two-hour intervals until 10 a. m. Dec. 16, 1953, and, finally, at three-hour intervals until discharge from the hospital Dec. 17, 1953. A total of 22 injections of Adrenosem were given. The patient was given 5 mg. of Adrenosem per oram every four hours day and night for two additional days after discharge from the hospital.

The nasal bleeding was reduced to spotting within two hours after therapy was started, and the spotting stopped between noon and 3 p. m. Dec. 15, 1953. It recurred briefly at 6 p. m. Dec. 15, 1953, but it did not recur thereafter.

The blood pressure on admission to the hospital was 230/150. It was taken four times daily, and on discharge from the hospital it was 210/150.

Laboratory studies made Dec. 15, 1953, showed that the erythrocyte count was 4,370,000 and the value for hemoglobin was 13.5 gm. There were 7550 white blood cells, with 75% segmented cells, 23% lymphocytes, and 2% eosinophiles.

The patient was seen again in the accident dispensary of the hospital at 8 a. m. March 5, 1954, with a history of bleeding from the right nostril at intervals since 7 a. m. the preceding day. He had secured some Adrenosem tablets from a drug store and took them, without completely controlling the bleeding, which became profuse a few minutes before his coming to the hospital. Examination revealed profuse bleeding from the right choanal region.

One injection of Adrenosem was given in the accident dispensary, and the patient was hospitalized. Injections were continued at half-hour intervals until the bleeding finally stopped at 1:30 p. m. March 5, 1954. They were then given at one-hour intervals day and night until discharge from the hospital March 7, 1954. A total of 48 injections were given, with per oram therapy continued at home as was done previously.

The blood pressure on admission was 170/130. It was recorded at hourly intervals day and night during the entire hospital stay. The peak pressure recorded at any one time was 240/140. On discharge from the hospital it was 150/100.

Laboratory studies made March 5, 1954, revealed an erythrocyte count of 4,270,000 and a hemoglobin value of 13.5 gm. There were 10,500 white blood cells, with 83% segmented cells, 15% lymphocytes, and 2% eosinophiles.

CASE 9.—N. L. S., a Negro man, aged 41 years, referred by Dr. E. P. Ewers, was first seen in the office Dec. 16, 1953. The patient had been bleeding from the right nostril at intervals for two days. The referring physician determined that the bleeding was coming from the right choanal region, but he had been unable to stop it by Gelfoam and cotton pack. He stated that the patient had been under his care for a moderate hypertension for some time. Fresh blood was present in the nose and throat, with bleeding from the back of the nose on the right side, when the patient was first examined in the office.

One injection of Adrenosem was given in the office, and the patient was then hospitalized. Injections were then continued at one-hour intervals for five injections; then at two-hour intervals for five injections; finally, at four-hour intervals until 10:30 p. m. Dec. 18, 1953. At this time the patient coughed and bled freely from the right nostril for several minutes. Adrenosem was then given at one-hour intervals until discharge from the hospital in the morning of Dec. 19, 1953. A total of 28 injections of Adrenosem were given. Per oram therapy was continued at home, 5 mg. of Adrenosem being taken very four hours day and night for three days.

The blood pressure on admission was 180/120. It was taken at hourly intervals, and the peak pressure recorded at any time was 190/130. The final recording on discharge from the hospital was 150/90.

The results of laboratory determinations made Dec. 17, 1953, showed an erythrocyte count of 4,070,000 and a hemoglobin value of 13 gm. The white blood cells totalled 8800, with 73% segmented cells and 23% lymphocytes.

CASE 10.—M. H., a white woman, aged 49 years, was first seen in the office on the morning of Dec. 29, 1953, with a history of bleeding from the right nostril at intervals, but with increasing tempo since the night of Dec. 28, 1953. She had a mild head cold of several days' duration. The patient exhibited marked apprehension to the extent that she was hardly able to talk.

Examination in the office revealed a generalized nasal congestion, with mild bleeding from the right choanal region. One injection of Adrenosem was given at hourly intervals in the office for a total of three injections, and then 5-mg. tablets were prescribed to be taken at hourly intervals at home. The patient returned to the office at 4 p. m. on the same day with a history of two bleeding spells during the afternoon. There was no bleeding at this time.

The patient was hospitalized at 6 p. m. Dec. 29, 1953, and Adrenosem administered by injection at hourly intervals day and night for 24 hours. It was then given at two-hour intervals day and night until discharge from the hospital Jan. 2, 1954. A total of 45 injections of Adrenosem were given. For two days after discharge from the hospital the patient took 5 mg. of Adrenosem every four hours day and night.

After hospitalization spotting of blood from the right nostril continued until 11 a. m. Dec. 30, 1953. It then ceased but recurred briefly at 6:30 a. m. Jan. 1, 1954. There was no further bleeding after this time.

The blood pressure on admission was 150/100. It was taken at hourly intervals day and night. The peak pressure recorded at any time was 160/100, and the final pressure on discharge from the hospital was 120/70.

The results of laboratory examinations made Dec. 30, 1953, showed an erythrocyte count of 3,620,000 and a hemoglobin value of 12 gm. There were 5900 white blood cells, with 65% segmented cells and 35% lymphocytes.

CASE 11.—S. J., a Negro woman, aged 37 years, referred by Dr. G. Smith, was first seen in the office Jan. 3, 1954. The right nostril had been bleeding at intervals for three days. A physician was consulted at the onset, and he prescribed tablets, which were without benefit. At 6 a. m. Jan. 3, 1954, the right nostril began to bleed freely. The patient then consulted the referring physician, who was unable to control the profuse bleeding. Examination in the office showed both nostrils to be filled with fresh blood clots, which were removed. Bleeding occurred from the back of the nose on the right side. One injection of Adrenosem was given in the office and the patient hospitalized.

Injections of Adrenosem were given at hourly intervals for three times and then at two-hour intervals day and night until the patient's discharge from the hospital at 2 p. m. Jan. 4, 1954. A total of 13 injections were given.

Bleeding from the nose stopped soon after her admission to the hospital. It recurred briefly at 1:30 a. m. Jan. 4, 1954, but not thereafter. The patient took 5 mg. of Adrenosem by mouth every four hours day and night for two days subsequent to discharge from the hospital.

The blood pressure on admission to the hospital was 170/110. It was recorded hourly day and night. The peak pressure recorded was that on admission. The blood pressure on discharge from the hospital was 120/70.

Laboratory studies made Jan. 4, 1954, showed a total of 3,760,000 red blood cells and a hemoglobin value of 12 gm. There were 5400 white blood cells, with 46% segmented cells, 53% lymphocytes, and 1% eosinophiles.

CASE 12.—W. H. M., a white man, aged 73 years, referred by Dr. K. P. Turrentine, was first seen in the hospital Feb. 7, 1954. The referring physician submitted a diagnosis of cardiovascular renal sclerosis with renal insufficiency and hypertension. The patient was chronically and critically ill. He had been hospitalized one week before for general medical care. During this time he developed brief episodes of bleeding from the left nostril, with spontaneous arrest. He had been subject to mild nose bleeds and crusting in the nasal cavities for many years. Two hours before admission Feb. 7, 1954, the patient began to bleed freely from the left nostril, and he was hospitalized for control of the hemorrhage.

Examination in the hospital several hours after admission revealed extensive crusting in both nasal cavities, with old blood clot present in both. A fresh blood clot was noted on the pharynx. The patient had been bleeding freely from both nostrils on admission to the hospital. Removal of the clots resulted in bleeding from the left choanal region, which subsided in about 30 minutes. The nasal septum was deviated markedly to the right, and there was a marked pallor of the nasal mucosa.

Adrenosem was given every 30 minutes for four injections, then hourly day and night until Feb. 8, 1954, and thereafter every two hours day and night until Feb. 12, 1954. Injections were stopped on this date, and 5 mg. of Adrenosem per oram was given four times daily through Feb. 17, 1954. A total of 93 injections were given.

The patient bled briefly in the early morning of Feb. 9, 1954, and Feb. 13, 1954, without recurrence after the latter date.

The blood pressure on admission was 190/80. It was taken at hourly intervals. The peak pressure recorded at any one time was 220/118, and the last blood pressure recorded was 176/82.

Laboratory studies made Feb. 9, 1954, showed that the erythrocyte count was 2,970,000 and the value for hemoglobin was 8 gm. On Feb. 19, 1954, the erythrocyte count was 2,500,000 and the value for hemoglobin was 9.5 gm. The blood urea nitrogen was 65 mg/100 cc.

CASE 13.—E. D., a Negro girl, aged 9 years, referred by Dr. J. Dawson, was first seen in the hospital March 5, 1954. The grandmother stated that the child had been bleeding from the left nostril for brief intervals over a period of two weeks. She had not seemed well for the preceding three months. The referring physician saw the patient at home at 1:30 a. m. March 4, 1954, and packed the left side of her nose. The bleeding recurred in the early morning of March 5, 1954, and hospitalization was advised.

On admission to the hospital there was no active bleeding from the nose. Because of the uncooperative nature of the patient, all available personnel were required to hold the patient while the nasal packing was removed. Bleeding then occurred briefly from the left Kiesselbach's area. It could not be determined whether this was the original site of hemorrhage or whether this area was traumatized by insertion or removal of the pack.

The rectal temperature on admission was 102 F. It ranged from 101 to 102 F throughout the entire hospitalization, and no satisfactory explanation was ever found to fully account for the fever.

On admission the erythrocyte count was 1,980,000 and the value for hemoglobin was 27%. The leuco-

cyte count was 14,400, with 81% segmented forms, 13% lymphocytes, and 6% eosinophiles. A note was made that there was a marked loss of hematin. Examination of the urine March 6, 1954, showed 18 to 20 white blood cells per high-power field. On March 8, 1954, the bleeding time was reported as 2 minutes and 35 seconds and the blood coagulation time as 4 minutes and 5 seconds. No sickle cells were present in the blood smears. Stool studies proved to be negative for ova or parasites.

The blood pressure on admission was 90/60. It peaked at 100/60.

The patient was seen in consultation March 5, 1954, by Drs. W. E. Keiter and J. Arnold, who advised correction of the anemia with hematimics rather than blood transfusions unless the latter were necessary to aid in the control of active nasal bleeding.

On admission to the hospital March 5, 1954, Adrenosem was given at 30-minute intervals for three injections and at hourly intervals thereafter for five additional injections. At 10 p. m. March 6, 1954, the patient bled from the back of the nose on the left side at intervals for two hours. I was with the patient during this time and estimated the blood loss at about 1 to 2 oz. The bleeding was more in the nature of periodic spotting. Adrenosem was now given at hourly intervals day and night for a total of 26 injections. Bleeding occurred briefly from the left nostril at 2 a. m. March 7, 1954. There was no further bleeding until 7:50 p. m. and 10 p. m. March 10, 1954, when brief episodes of spotting occurred from the right nostril. Adrenosem was started at 10 p. m. and given at hourly intervals for 11 injections. On March 12, 1954, from 2:30 a. m., the patient bled briefly at intervals until 6:30 a. m., the bleeding being more in the nature of periodic spotting. The total number of injections of Adrenosem was 45.

During the first two days of hospital admission it was noted that the patient's sensorium appeared to be somewhat cloudy at times. After these intervals she was mentally alert, read comic books, ate, and took prescribed medication well. At 10 p. m. March 7, 1954, the patient suddenly became mentally confused and developed generalized tremors, with a rapid thready pulse. She was seen at this time by Dr. Arnold and me. The attack was passing spontaneously as we arrived at the hospital. At 7:30 a. m. March 12, 1954, the patient developed a similar attack, at which time she was seen by Dr. Arnold. Intranasal oxygen was started along with a transfusion of whole blood. The patient recovered from the attack but died a short while later of a similar type of attack.

The management of this case was hampered by the totally uncooperative nature of the patient. After blood was secured for the first laboratory studies, neither the laboratory personnel nor Dr. Arnold was ever able to secure more blood for study. Examinations were conducted with difficulty and incompletely because of the patient's belligerent nature. Certain interesting features of this case not germane to the present topic have not been discussed.

Case 14.—F. D., a white man, aged 47 years, referred by Dr. R. H. Temple, was first seen in the hospital March 9, 1954. Early in the morning of the same day the patient developed sudden profuse bleeding from both nostrils. The referring physician saw the patient in his office and stated that the bleeding was coming from the back of the nose, although he was unable to determine from which side. He inserted anterior nasal packs at this time. Profuse bleeding again occurred from both nostrils at 7 p. m., and the patient was hospitalized. The patient had been subject to frequent head colds all winter, with blood streaking in the nasal discharge, and recently he had a severe head cold. I saw the patient about three hours later and removed the nasal packs. There was congestion of the nasal mucosa, and the nasal septum was deviated to the right, with considerable obstruction. No fresh blood was in evidence at this time.

Adrenosem was given hourly for 8 injections and then every two hours day and night for an additional 14 injections. A total of 22 injections were given. The patient took 5 mg. of Adrenosem every four hours day and night for two days after discharge from the hospital.

The patient stopped bleeding soon after admission to the hospital. He bled again briefly at noon March 10, 1954, but not thereafter.

The blood pressure on admission was 140/80, and it did not exceed this during the hospital stay. Blood pressure was recorded at hourly intervals, and the final recording was 110/70.

Laboratory studies made March 10, 1954, showed a total of 4,400,000 red blood cells and a hemoglobin value of 13 gm. There were 12,300 white blood cells, with 61% segmented cells, 36% lymphocytes, and 3% eosinophiles.

Case 15.—Z. K., a white woman, aged 84 years, was first seen in the office March 19, 1954, with a history of bleeding from the left nostril during the past 24 hours. Examination revealed that the patient was bleeding from the back of the nose on the left side. She was given Adrenosem every 30 minutes for a total of four injections, at the end of which time the bleeding had stopped. Five milligrams of Adrenosem every four hours day and night was prescribed for home use.

The patient returned to the office at 9 a. m. March 20, 1954, stating that she had a recurrence of the bleeding in the early morning. The bleeding originated from the back of the nose on the left side. It was profuse, and the patient was hospitalized.

Adrenosem was given at hourly intervals for a total of 16 injections and then continued at two-hour intervals for an additional 14 injections, making a total of 30 injections.

The patient bled (spotting) until noon on the day of admission, without recurrence thereafter.

The blood pressure on admission was 130/70. The final recording was 124/70.

The patient was seen in consultation by Dr. W. T. Parrott Jr., who rendered a diagnosis of generalized arteriosclerosis, senility, and senile-type emphysema.

On the day following admission the patient developed an acute nasopharyngitis, which prolonged the period of hospitalization.

Laboratory determinations made March 20, 1954, showed a total erythrocyte count of 4,640,000 and a value of 13 gm. for hemoglobin. There were 9400 white blood cells, with 79% segmented cells and 21% lymphocytes.

Case 16.—S. L. K., a white man, aged 65 years, referred by Dr. G. Smith, was first seen in the office May 11, 1954, with a history of bleeding from the right nostril at intervals for three weeks. The patient gave a history of long-standing hypertension. The referring physician had been unable to stop the hemorrhage by either local or general measures and referred the patient when the hemorrhage became profuse. When examination was made in the office, blood was flowing freely from the anterior nares and dripping off the uvula in a steady drip. The nasal septum showed marked deviation to the right anteriorly. The bleeding was coming from the right nostril posterior to the obstructing portion of the septum. One injection of Adrenosem was given in the office, and the patient was hospitalized.

Adrenosem injections were given at hourly intervals day and night for a total of 22 injections, then at two-hour intervals for 14 additional injections, and, finally, at three-hour intervals for 4 injections. A total of 40 injections of Adrenosem were given. The patient took 5 mg. of Adrenosem every four hours day and night for four days subsequent to discharge from the hospital.

The nasal bleeding stopped in four hours after his admission to the hospital. It recurred briefly at 5 p. m. May 13, 1954, and finally in the early morning of May 14, 1954.

The blood pressure on admission was 170/96, and this was the only recording made on the chart.

The patient was seen in consultation by Dr. L. E. Williams, who made a diagnosis of hypertensive cardiovascular disease with arterial sclerosis.

The results of laboratory studies made May 12, 1954, showed an erythrocyte count of 4,580,000 and a hemoglobin value of 14 gm. There were 7750 white blood cells, with 79% segmented cells, 20% lymphocytes, and 1% eosinophiles.

Case 17.—B. E. H., a white man, aged 51 years, was first seen in the office May 12, 1954, with a history of intermittent bleeding from the right nostril for three days. About half an hour previously the bleeding became profuse. Three weeks before, the patient had been confined to his bed at home because of a severe respiratory infection. He gave a history of severe nose bleed seven years previously, at which time his nose was packed, and a number of blood transfusions were given during a prolonged period of hospitalization. At this time the patient stated that he was told by attending physicians that he had "liver trouble" and "kidney stones." The patient was quite apprehensive lest the present episode of bleeding follow the previous course. The patient was given one injection of Adrenosem in the office and hospitalized.

Adrenosem was given at one-hour intervals for a total of 88 injections, then at two-hour intervals for an additional 12 injections, and, finally, at three-hour intervals for an additional 22 injections. A total of 122 injections were given. The patient was given 5 mg. of Adrenosem to take by mouth every four hours day and night for three days after returning home.

The nasal bleeding stopped in four hours after admission to the hospital. There were brief episodes of bleeding in the early morning and on the night of May 14, 1954, and in the afternoon of May 16, 1954. There was no recurrence after the latter date.

The blood pressure on admission was 170/96, and on the following day it was 120/80.

On May 14, 1954, urinalysis showed that the urine was loaded with blood with 6 to 7 white blood cells per high-power field and occasional granular casts. The blood urea nitrogen was 28 mg/100 cc. The sedimentation rate was 18 mm. in 30 minutes and 43 mm. in 60 minutes On May 18, 1954, there were 20 to 30 red blood cells per high-power field in the urine.

A survey film of the abdomen made May 14, 1954, showed shadows suggestive of calculi in the left urinary tract.

The patient was seen in consultation by Dr. K. P. Turrentine.

Case 18.—H. L. C., a white woman, aged 45 years, referred by Dr. V. D. Offutt, was first seen in the office Oct. 9, 1953, with a history of bleeding from the left nostril since 1:30 a. m. Oct. 9, 1953. Packing of the left nostril by the referring physician failed to control the bleeding. The patient had been a known hypertensive for 18 years.

Examination in the office showed that the patient was bleeding freely from the left lateral nasal wall in the region of the middle third of the middle meatus. All efforts to control the bleeding in the

office were futile. She was hospitalized for insertion of a postnasal pack, which was left in situ for five days and then removed.

Laboratory studies made Oct. 10, 1953, revealed an erythrocyte count of 4,520,000 and a hemoglobin value of 13 gm. There were 13,700 white blood cells, with 62% segmented cells, 36% lymphocytes, 1% monocytes, and 1% eosinophiles.

The patient returned to the office on the morning of Feb. 19, 1954, with a history of bleeding from the right nostril intermittently since 4 a. m. A short while before the patient was seen in the office, the bleeding had become constant and profuse. The patient had a head cold at the time. Examination in the office revealed that the bleeding originated from the right lateral nasal wall in the region of the middle third of the inferior meatus.

Adrenosem was given in the office at intervals of 30 minutes for a total of four injections. The patient then took 5 mg. of Adrenosem by mouth every four hours day and night for two days.

Bleeding from the nose stopped in 30 minutes after the first injection of Adrenosem and did not recur.

CASE 19.—C. G., a Negro man, aged 23 years, was first seen in the office July 3, 1954, with a history of intermittent bleeding from the left nostril for three days. During the first two days of bleeding the patient used home remedies in a futile effort to stop the bleeding spells. He then consulted his family physician, who packed the left nostril with cotton on the morning of the third day of bleeding. This also failed to control the bleeding, and the patient was seen in the office about three hours later.

Examination in the office revealed active bleeding from the left choanal region of the nasal cavity, which was beyond control. The patient was hospitalized for Adrenosem therapy.

Two cubic centimeters of Adrenosem was given on admission to the hospital and repeated at intervals of one hour for a total of three injections. Active bleeding from the nose stopped within 2 hours and 30 minutes from the first injection of Adrenosem. On July 4, 1954, the patient was started on 5 mg. of Adrenosem per oram every four hours day and night. At 1 a. m. July 5, 1954, the nasal bleeding returned, and 1 cc. of Adrenosem was given at intervals of one hour for a total of three injections. The bleeding stopped but recurred at 5 p. m. July 5, 1954. Adrenosem injections were now started and continued at intervals of one hour for a total of 52 injections. The nose continued to bleed briefly at intervals until 4:30 p. m. July 6, 1954, after which time there was no further hemorrhage.

The blood pressure on admission was 110/60. It was charted at intervals of one hour as long as Adrenosem was given by injection and q. i. d. during the remainder of the hospital stay. The peak pressure attained at any time was 150/70. The final recording was 120/60.

Laboratory studies made July 5, 1954, showed a red cell count of 2,360,000 and a hemoglobin value of 7 gm. There were 10,050 white blood cells, with 87% segmented cells, 12% lymphocytes, and 1% monocytes.

The patient was seen in consultation by Dr. C. Wooten, who gave a transfusion of 500 cc. of whole blood July 9, 1954, three days after nasal bleeding had stopped completely.

CASE 20.—E. K., a Negro man, aged 48 years, was first seen in the office Aug. 17, 1954, with a history of bleeding from the left nostril throughout the day. The patient consulted his family physician, who gave him three injections and some tablets to take, which were without effect in controlling the bleeding.

Examination in the office revealed active bleeding from the choanal region of the nose on the left side. The bleeding could not be controlled, and the patient was hospitalized for Adrenosem therapy.

One cubic centimeter of Adrenosem was given on admission to the hospital, and this was repeated at intervals of one hour for a total of 21 injections.

Active bleeding from the nose stopped within one hour after the first injection of Adrenosem and did not recur.

The blood pressure was 190/120 on admission to the hospital, and the peak pressure recorded at any time did not exceed the pressure on admission.

Laboratory studies made Aug. 18, 1954, showed an erythrocyte count of 3,790,000 and a hemoglobin value of 10 gm. There were 8100 white blood cells, with 78% segmented cells and 22% lymphocytes.

MISCELLANEOUS CASES

Traumatic Hemorrhage from the Larynx

CASE 21.—F. W., a Negro man, aged 45 years, referred by Dr. C. Wooten, was first seen in the office Feb. 20, 1954, with a history of having been kicked in the neck by assailants the night before. In addition to other laryngeal symptoms, the patient had been coughing up red blood since the accident. Examination revealed a traumatic laceration of the right side of the larynx involving the aryepiglottic fold and right arytenoid cartilage. The bleeding was coming from this area. It was subsequently proved that the patient had a fracture dislocation of the right arytenoid cartilage. The tip of the vocal process had been completely separated from the arytenoid cartilage, and this was later removed from the lacerated area as a foreign body.

The patient was hospitalized and Adrenosem given by injection at intervals of 30 minutes for a total of four injections, at the end of which time the bleeding had stopped permanently.

Laboratory studies made Feb. 20, 1954, showed an erythrocyte count of 4,430,000 and a hemoglobin value of 14.5 gm. There were 20,540 white blood cells, with 73% segmented cells, 24% lymphocytes, and 3% monocytes.

Hemorrhage from a Tonsillar Crypt

CASE 22.—W. G., a white boy, aged 3 years, referred by Dr. W. E. Keiter, was first seen in the office April 21, 1954. The patient had measles. On the preceding night and again on the morning of April 21, 1954, the patient spit up red blood, without cough or bleeding from the nose. The first examination of the nose, nasopharynx, and larynx failed to reveal any old or fresh blood or bleeding point. As the tongue was depressed for a second examination, blood spurted out of a deep cleft in the upper pole of the left tonsil and ran freely into the pharynx in a steady stream.

The patient was given one injection of Adrenosem, and the bleeding stopped permanently in about 20 minutes. Five milligrams of Adenosem was then given by mouth every four hours day and night for two days.

COMMENT

A definite impression has been formed that the preoperative use of Adrenosem has decreased the bleeding attending the removal of tonsils and adenoids at the time of operation and has caused a diminution in the amount of bloody oozing from the nose and mouth in the immediate postoperative period. The oozing from the nose seen in cases of submucous resection of the nasal septum a few hours after completion of operation has been a source of annoyance to patients, although to date the bleeding fortunately has not been of such magnitude as to warrant local postoperative intervention in any of my cases. The use of Adrenosem has reduced this oozing to a minimum. It has also been instrumental in reducing the bleeding following removal of the nasal packing the next day.

This impression has been shared by operating room assistants, anesthetists, and floor nurses as well as by me. Unfortunately, it is difficult to provide tangible evidence to support this opinion.

There are some tangible data recorded in this study that can be discussed in terms of a preliminary report. The average operating time for 313 primary and secondary tonsillectomies and adenoidectomies performed on both children and adults before the routine use of preoperative Adrenosem was computed and found to be 19.99 minutes. The average operating time in 176 similar type operations on similar type patients who received preoperative Adrenosem was computed and found to be 17.39 minutes. The specific details are recorded in Tables 2 and 3. It is known that factors other than bleeding may influence operating time. However, it seems acceptable to consider that the facts to date indicate a trend toward shortened operating time due to diminished bleeding from the preoperative use of Adrenosem.

The records of 336 tonsillectomies and adenoidectomies performed from Jan. 1, 1952, through Nov. 19, 1953, before the preoperative use of Adrenosem, were reviewed to determine the incidence of patients requiring suture of the tonsillar fossae to control bleeding at the time of operation. It was found that 28 out of 336 patients required sutures (Table 2). This affords an incidence of 8.3% of patients who required sutures. In this same group of 336 tonsillectomies and adenoidectomies, there were 13 cases of primary and secondary postoperative hemorrhages (Table 4), with 9 of these being the primary type. This gives an incidence of 2.6% of patients with the primary type of postoperative hemorrhage.

Out of 176 patients who received preoperative Adrenosem, 6 required sutures for control of bleeding from the tonsillar fossae at the time of operation (Table 3). This gives an incidence of 3.4% of Adrenosem-treated patients who required sutures at time of operation to control bleeding from the tonsillar fossae. In this same group of 176 patients, there were 7 cases of postoperative hemorrhage, 2 cases each of primary adenoid and tonsillar hemorrhage, and 3 cases of secondary adenoid bleeding (Table 5). The incidence of primary postoperative hemorrhage in this group is 2.2%. These figures indicate a definite trend toward fewer sutures and perhaps fewer cases of primary postoperative hemorrhage.

In the seven cases of primary and secondary postoperative hemorrhage which were

treated with Adrenosem, the clot was removed outside the operating room in five instances and in the operating room in the remaining two instances. An average of four to six injections of Adrenosem were given to control the bleeding. Postnasal packing was not necessary. While it is recognized that removal of the clot may be sufficient to arrest postoperative hemorrhage in some cases, it is my opinion that none of these seven cases would have responded to this treatment alone. The results indicate a simpler control of postoperative tonsillar and adenoid bleeding by Adrenosem therapy.

No definite trends can be set forth regarding the use of Adrenosem in laryngologic, bronchoscopic, and esophagoscopic procedures, with one exception. The bleeding that sometimes follows the removal of benign inflammatory growths from the vocal cords hampers vision and inspection of the cords immediately after removal of the growth. In my experience this has been particularly true in the cases of subglottic growths. It is my impression from a small number of cases that the preoperative use of Adrenosem reduces this bleeding and improves visualization and inspection of the pathologic area after removal of this type of growth.

The control of epistaxis has been far more troublesome in my practice than the control of postoperative surgical bleeding. It has been my custom, and still is, to stop all accessible bleeding points on the nasal septum by submucosal injection of procaine followed by cauterization with the actual current. This has been a highly successful procedure in my hands. A preliminary injection of Adrenosem 20 to 30 minutes before injection of the procaine slows up the bleeding and thus facilitates cauterization. Oxidized cellulose (Hemo-Pak) has been of inestimable value to me in the control of active bleeding from the nasal septum in children and in the control of bleeding from the anterior third of the lateral nasal wall in the regions of the inferior and middle meatuses in adults.

Hemorrhage from the posterior region of the nasal septum or lateral nasal wall has always been a troublesome problem in my practice. In all instances of bleeding from these areas I have in the past exhausted all possible means of local control before resorting to a postnasal pack, but in only one case since 1948 have I been able to arrest hemorrhage by means other than postnasal pack until the advent of Adrenosem. In two instances before the use of Adrenosem repeated postnasal packing failed to control the bleeding in arteriosclerotic persons, and ligation of the external carotid artery was necessary.

The results with the use of Adrenosem in the control of bleeding from the back of the nose indicate that it is a valuable ally in the treatment of this condition. Since there apparently has been very little published regarding the use of Adrenosem in the control of epistaxis, my own efforts to establish the necessary therapeutic amount of Adrenosem was largely on a trial and error basis. It was felt that the most satisfactory plan of procedure would be to establish the maximum dosage necessary and to reduce this as experience dictated. It is felt, therefore, that the dosage as set forth in the case reports represents maximum therapy and that some of these cases might well have done with fewer injections of Adrenosem.

In several patients with epistaxis the blood pressure was recorded at hourly intervals during the course of Adrenosem therapy. As was to be expected, there was no case that showed evidence of increase in blood pressure as a result of Adrenosem action. As a matter of fact, no untoward effect of any type was noted in the Adrenosem-treated group.

One case of epistaxis treated with Adrenosem has been omitted from the case histories. This patient received additional therapy ordered by the consulting physician before this was brought to my attention. The nose bleed was checked, and the drugs used in concomitant therapy are listed in Table 6 for the "Unrecorded case."

It seems likely that the present plan of preoperative Adrenosem therapy is adequate. Efforts are now being directed to reduce the

number of injections used in the treatment of bleeding from the back of the nose. It should be remembered that all the cases of epistaxis recorded in this report are cases which, in my opinion, would have required insertion of a postnasal pack for control of the bleeding had it not been for Adrenosem therapy. At the present moment it appears that most cases of bleeding from the choanal region of the nose must be hospitalized for Adrenosem therapy. In severe cases it may be well to give Adrenosem at intervals of one hour for the first 24 hours and then increase the intervals of injection as the case seems to warrant.

CONCLUSIONS

The data assembled in this study seem to warrant the following preliminary conclusions. The routine preoperative use of Adrenosem in tonsil and adenoid surgery diminishes both the bleeding at the time of operation and the oozing in the immediate postoperative period. As a result of the reduced bleeding at operation fewer patients require suture of the tonsillar fossae for the control of hemorrhage. The incidence of primary postoperative hemorrhage may be reduced. Adrenosem is valuable in the active treatment of postoperative tonsil and adenoid bleeding and may reduce the incidence or obviate the necessity of postnasal packing or sutures.

In cases of submucous resection of the nasal septum the use of Adrenosem reduces the bleeding at the time of operation, in the postoperative period, and after removal of the nasal packing.

Preoperative Adrenosem reduces the bleeding following removal of benign inflammatory growths from the subglottic larynx, and the operative field is thereby better visualized to determine completeness of removal.

Adrenosem is valuable in the control of hemorrhage from the choanal region of the nose seen in cases of hypertension, arteriosclerosis, or respiratory infections with or without local predisposing pathological conditions in the nasal cavity. The necessity of postnasal packing may be obviated by Adrenosem therapy in the majority of these types of cases.

The amount of Adrenosem employed in all the case reports represents the maximum amount necessary to obtain desired therapeutic results.

Adrenosem does not produce undesirable side-effects when used in the amount and at the intervals employed in this group of cases.

The Administrator of Lenoir Memorial Hospital made Adrenosem available for use in ward cases.

Kinston Clinic.

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Lymphosarcoma of the Tonsil

A Case Report and a Brief Review

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Malignant growth of the tonsil is seen relatively infrequently by the great majority of physicians; yet it is of interest because (*a*) discovery of a case is provocative of a general review of this uncommon sarcoma; (*b*) the diagnosis and treatment of this neoplasm is established and followed very early in the course of this disease; (*c*) the statistical possibilities of cure are encouraging if one does happen to encounter this very uncommon growth. In the literature the statistics vary considerably on tonsillar cancer. Telch [1] states that the five-year survival rate for all patients with tonsillar lymphosarcoma is 22%; with carcinoma, 5%. Ackerman and del Regato [2] report 40% five-year cures in a group of 37 cases of lymphosarcoma of the tonsil. Parshall and Stenstrom [3] report 6 (46%) survivals for three years among 13 patients and 3 (30%) among 10 for five years. This latter report was, of course, on a small group of lymphosarcomata of the tonsil. The Curie Foundation [4] reports 34 (33%) cases of five-year survivals out of 104 cases of tonsillar lymphosarcoma. Differences would naturally occur in these statistics, however, as some are derived from the surgical literature and others from the radiological literature, but the above statistics do compare very favorably with the usual cure rates for carcinoma of the breast, which is very dependent upon the important question of localization of the tumor in regard to its curability. A solitary failure, such as we report, must therefore not disrupt any thinking in regard to the proved statistical possibilities of cure.

Lymphosarcoma is unquestionably one of the most radiosensitive of all malignant neoplasms, but, despite its very satisfactory response to irradiation, the prognosis, as with other tumors, depends upon whether it remains localized or presents generalized involvement. There has been some controversy over the question of whether the lymphoblastomata are unicentric or multicentric in origin. Certainly the number of five-year survivals of lymphosarcoma of the tonsil as reported in the literature would indicate that in an appreciable precentage, at least, the disease arises from a single focus and can frequently be controlled. Thus, optimism should be present at the discovery of a case, not undue pessimism, as one might experience from a solitary failure.

May we emphasize that cervical lymph node metastasis is an important factor in determining the ultimate result. Early detection, with treatment, both surgical and radiological, is imperative. The usual symptoms with tonsil cancer are a mass in the pharynx, sore throat, pain referred to the ear, jaw, occiput, and temporal regions, with a subsequent course of dysphagia, weight loss, sore mouth, and early cervical adenopathy and, later, systemic manifestations of metastases. The lesion is visible, palpable, and indurated. Systemic lymphoid areas must be examined for resultant splenomegaly, hepatomegaly, generalized adenopathy, pleural and abdominal ascites, and lymphedema. The lesion may be visualized as nodular or ulcerative or, as occurred in this case, as a large globular mass present in the tonsillar fossa, which is at first thought of as a tonsillar abscess, secondarily as a possible tumor mass. It may involve the anterior and posterior tonsil pillars, and when it extends toward the oropharynx it causes dysphagia. The following observations and comments are a very condensed

form of the most pertinent clinical findings, course, and treatment of a singular case of lymphosarcoma of the tonsil.

REPORT OF CASE

Mrs. Otto B., aged 55, a very cooperative and intelligent patient, had been seen by us over a period of four years for menopausal symptoms and obesity. She had no previous contributory illnesses or pertinent medical record, with the exception of having had three miscarriages and three living children during her lifetime. Physical findings were negative when she was first examined in 1949, except for aortic regurgitation and bilateral hypertrophic tonsils. Because of this marked tonsillar hypertrophy, tonsillectomy was advised but not agreed to by her. In retrospect, more persuasion should have been expended relative to a tonsillectomy. Her menopausal symptoms were controlled by periodic estrogen therapy and the obesity controlled by diet, over a period of four years. After this, she felt well and was not seen by us for four months, from August to November, 1951. On Nov. 20, 1951, she presented complaints of a sore throat, present for the past week, with pain in the right lower jaw. There was a mass about 3 by 6 by 3 cm. in the right tonsillar fossa, clinically suggestive of the common tonsillar abscess. There was cervical adenopathy. Differential diagnosis consisted of early abscess or of tumor mass. She was given penicillin and dihydrostreptomycin intramuscularly on alternate days for the next 10 days, and on the sixth day the pointing, globular tonsil mass was incised and drained of purulent and necrotic debris. She was again advised to be hospitalized for the removal of an ulcerative, necrotic, gangrenous right tonsil, and agreed and submitted to surgery on Nov. 30, 1951, with the usual gas anesthetic, dissection, and snare procedure. The tonsillar mass was dissected free and intact from the pillars and fossa, and, since it felt indurated and was necrotic and ulcerative, it was sent to a pathologist for microscopic examination. He reported a reticulum-cell lymphosarcoma. Her immediate postoperative course was uneventful. She was advised to have deep radiation therapy to involved areas at once and did so intermittently during the next 11 months. The irradiation therapy was as follows: From Dec. 19, 1951, to Jan. 2, 1952, Mrs. B. was given 10 doses of 200 r (in air) to each of two opposing lateral 10 by 10 cm. fields directed to the tonsillar areas and including the uppermost cervical chain. Technical factors employed there were 250 kv., 30 ma., 50 cm. distance, 0.5 mm. Cu filter with 1.0 mm. Al added (1.50 mm. Cu HVL). Three months later (after it became obvious that her disease was generalized), smaller courses were administered to the right axilla, both inguinal areas, and the left epitrochlear region (six doses of 150 r [in air] with same factors). About two months after that, the spleen and the left axilla were given similar courses (also 6 doses of 150 r each, with the above factors). In August, 1952, five 200 r (in air) doses were given to each of two opposing anteroposterior fields over the left lower thorax and splenic area. A 1.0 mm. Cu filter with 1.0 mm. Al added was used (1.90 mm. Cu HVL). The other factors were the same. In November, six 150 r (in air) doses were given to each of these last-named portals with use of a Thoraeus filter, giving 2.75 mm. Cu HVL. After general metastases became evident, none of the areas treated showed the satisfactory response obtained in the primary site, even though temporary regression of adenopathy did occur. This was somewhat unusual, in view of the usual radiosensitivity manifested by lymphosarcoma, despite the difference in dosage employed. Speculative biochemical factors were presumptive. On Aug. 31, 1952, she had irradiation sickness, secondary anemia, and generalized lymphosarcomatosis. She was hospitalized for transfusions and parenteral feedings because of nausea and anemia. The spleen was palpable to the level of the umbilicus and was hard, nodular, and tender. There were lymphoid masses in the groin, popliteal angle, and medial portion of the right leg. She was rehospitalized on Oct. 30, 1952, for generalized lymphosarcomatosis, with left massive pleural effusion and complicating secondary anemia. Thoracenteses produced 1000 cc. of bloody fluid, which relieved her pain and dyspnea. Transfusions accomplished further gain in strength, but generalized adenopathy and splenomegaly remained. Final hospital admission was on Dec. 10, 1952, for generalized pain, cachexia, emaciation, hepatomegaly, splenomegaly, abdominal ascites, secondary anemia, and fecal impaction. She was treated symptomatically, sent home by ambulance for terminal care, at the family's request, with only the necessary narcotic therapy as prescribed. She finally died Jan. 11, 1953, of terminal pulmonary edema and pneumonia, having lived about 14 months between probable onset and termination.

COMMENT

There was a lapse of about one week between probable onset of the malignant growth and presentation of complaint by the patient to the physician. There was a short lapse, of 10 days, between the discovery of the suspected tumor mass and its excision, yet metastasis was early, rapid, and fulminating. It is interesting that there was a complete regression of evidence of involvement of the primary site after surgical treatment

and irradiation therapy. At no time was there recurrent adenopathy in the cervical lymph nodes. The primary site was adequately controlled throughout the course of this case. Death occurred because of generalized metastasis, with its resultant cachexia and pneumonia, a phenomenon common to many cases of lymphosarcoma of the tonsil and other malignant tumors.

SUMMARY

A singular case of lymphosarcoma of the tonsil is presented, briefly reviewing the pertinent literature, diagnoses, and treatment of this uncommon pharyngeal tumor. The statistical possibilities of cure are encouraging, since lymphosarcoma is one of the most radiosensitive of malignant neoplasms.

The following gave technical aid and other assistance: Thomas Tallant, medical technician and manager, Barron Clinic, Barron, Wis.; Norman Makous, M.D., locum tenens, Barron Clinic; C. C. Kirkham, medical technician and manager, Lakeside Hospital, Rice Lake, Wis., and Ralph C. Frank, M.D., radiologist, Sacred Heart Hospital, Eau Claire, Wis.

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Case Reports

RETICULUM-CELL SARCOMA ARISING IN THE PARANASAL SINUSES

Report of Case

BENJAMIN H. SHUSTER, M.D.
and
ALLAN R. SHUSTER, M.D., Philadelphia

ALTHOUGH reticulum-cell sarcomas are not uncommon malignant tumors, their primary origin in the paranasal sinuses is infrequent. There are scattered reports throughout the literature of such lesions arising in the sinuses. However, the literature is somewhat confusing to review because the classification of these tumors has been a point of controversy among some pathologists. One school describes these tumors as arising from undifferentiated mesenchymal or reticulum cells and have given the tumor its name of reticulum-cell sarcoma. However, other pathologists list these as lymphosarcomas. To make the picture still more confused, some pathologists, such as Ehrich, state that there is truly no such thing as a lymphosarcoma. In reviewing case reports it is extremely difficult to appraise the number of cases because of these differences in nomenclature and because many cases are in the literature listed as merely sarcomas with no attempt to further classify them. The otolaryngologic literature has been particularly careless in these matters. The result of this has been that there is only a handful of cases reported clearly as being reticulum-cell sarcoma arising primarily in the paranasal sinuses.

We are reporting this case for the following reasons: (1) because of its infrequent, if not rare, occurrence; (2) to call attention to the controversy among the pathologists so clinical otolaryngologists should not get caught in the web of their confusion, and (3) to review our approach to the problem, our course of treatment, and the results obtained.

REPORT OF CASE

H. R., a 50-year-old white man employed as a dairy manager, was well until December, 1953, at which time he developed an upper respiratory infection which left him with a residual frontal sinusitis. One week after this arose, his frontal sinuses were irrigated. Thereafter his right eye began to swell. Only a minimal amount of pain was associated with this. In the middle of January some vague intranasal surgery was performed and the frontal sinuses were drained. The right eye continued to be swollen. It was at this time that the patient was referred to us.

When we first saw him, he showed an orbital cellulitis on the right side. X-rays brought by the patient showed a pansinusitis on the right, with some suggestion of bone destruction of the medial wall of the right orbit. He gave the additional history of having had some degree of obstruction to his breathing for some 18 months prior to that time. Family history, past history, and systemic review were otherwise essentially negative. He had some occasional diplopia. Physical examination showed only the orbital cellulitis and the presence of some irregular tissue in the right nostril which was difficult to evaluate because of the recent intranasal surgery done elsewhere. There was no lymphadenopathy, hepatomegaly, splenomegaly, or kidney enlargement. Other than a mild hypochromic anemia, hematologic, urine, and blood chemistry studies were all within normal limits.

On Feb. 1, 1954, an external frontoethmoid approach to the sinuses on the right side was utilized. The ethmoids and frontal sinus on the right side were filled with a granulomatous-appearing mass which had eroded the walls of the ethmoid sinus as well as the anterior wall of the sphenoid sinus. Both sphenoid sinuses were also filled with this tissue, which was removed. The dura of the anterior cranial fossa had been exposed by the disease process, and we proceeded to remove all the necrotic bone which was present around the opening. Much pus and polypoid tissue were present in addition to the granulomatous-appearing tissue. The entire area was packed with iodoform gauze brought out through the nose. The external opening was closed

with nonabsorbable surgical (silk) sutures. The patient experienced little or no postoperative difficulty, and all packing was removed by the fourth postoperative day. The eye receded to its normal position in a matter of two or three days. In the meantime the pathology report on the tissue came back as reticulum-cell sarcoma.

He was seen in consultation by Dr. Beizer, who confirmed our own physical findings and reported that the bone marrow was not involved by the reticulum-cell sarcoma. A chest x-ray revealed a suspicious lesion in the right lung field, but this later proved to be an artifact. It was decided then to give a combined course of local irradiation and a systemic cytotoxic drug. Accordingly over a period of time extending for one month a total dose of 3000 r each was given through a frontal port, a right lateral port, and a left lateral port by Dr. Smith at the Graduate Hospital. In addition he was given a total dose of 25 mg. of triethylene-melamine over a period of one month. Other therapy at this time consisted only of supportive measures. At the conclusion of this, however, a chest film revealed some slight irregularity of the lateral aspect of the left ninth rib which had not been previously present. Also a sharply demarcated soft tissue mass was seen in the left hilum, but its significance was not certain. When the patient was again seen at the end of April, it was obvious that there had been a great weight loss. He had anorexia and pain in the left chest and right shoulder. Again no lymphadenopathy or liver or spleen enlargement was noted. No significant changes in the hematologic picture were noted. In view of the clinical evidence of dissemination, a course of nitrogen mustard was advised. He chose to have this done closer to his home in Lancaster. Following the course of therapy in June, he showed a remarkable response. Clinically and symptomatically he appeared entirely well. However, in September he once again returned to his earlier state of poor health. At this time a large node was present in the right submandibular area, but none was found elsewhere nor was there any spleen or liver enlargement noted. He was then given a second course of nitrogen mustard therapy at the Lancaster Hospital, with some relief but nowhere near the previous dramatic response. Cortisone therapy was also given for about a month. His condition remained the same until the first week in November, when early one morning he developed convulsive seizures at home and then slipped into a comatose state. He was readmitted by his family physician to the St. Joseph's Hospital in Lancaster. With much understandable trepidation and while the patient was still in coma, a third course of nitrogen mustard was administered. He responded by coming out of his comatose state to one of alertness. At this time the liver, spleen, and possibly kidney were palpable. The patient is currently still in the hospital but in such a weakened condition that he is just about able to sit up in bed and feed himself but unable to ambulate. In short, he appears in a terminal state.

The clinical picture of illness has been present now for about one year. Its course may be considered a relatively rapid one in that the usual course runs for 18 months following the presentation of symptoms. This point, however, merely reemphasizes the fact that malignant tumors of the paranasal sinuses are rarely found in their early stages but nearly always after there has been considerable growth. In our case there is finally a widespread invasion as well as recurrence of the growth locally, as indicated by the recent convulsions and comatose state. Five-year survivals from reticulum-cell sarcoma are indeed rare, although Towson [1] has recently reported a five-year survival of a case in which the lesion was in the nasopharynx. It is our feeling that in view of the highly malignant character of these lesions, the best treatment is a combination of palliative but nondeforming surgery, intense irradiation, and use of cytotoxic drugs to control dissemination.

1824 Pine St. (3).

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LEIOMYOMA OF THE TRACHEA

A Case Report

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SMOOTH muscle tumors are very rarely encountered in the upper respiratory tract. Eggston and Wolff,[1] in their textbook "Histopathology of the Ear, Nose and Throat," state that a few cases of leiomyoma in the esophagus and trachea have been reported; however, I was unable to find a single case of leiomyoma of the trachea recorded in the literature.

Several theories have been advanced as to the actual origin of leiomyomata. It seems probable that they arise from smooth muscle itself, but there are those who believe them to originate from the musculature of blood-vessel walls.[2]

Microscopically, leiomyomata are dense, hard masses which on section exhibit cystic spaces. The latter result from disintegration of the tissue due to the scarcity of blood vessels.

Microscopically, leiomyomata are composed of smooth muscle fibers arranged in parallel rows. Bundles interlace in every direction. These bundles are embedded in an abundant stroma of connective tissue, which bears the blood vessels. The muscle fibers are recognizable by their rod-shaped nuclei and by their relatively plump cell body.[2]

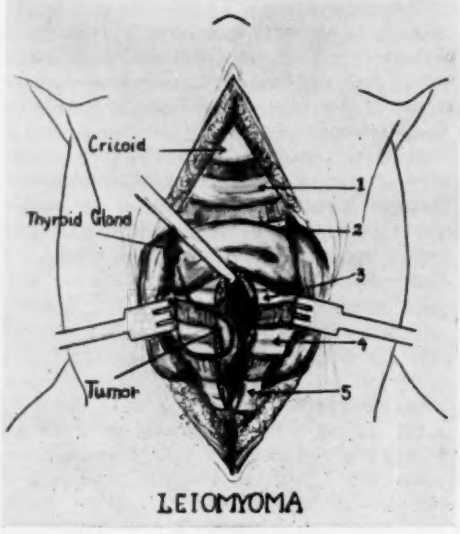
REPORT OF CASE

W. D. F., a 45-year-old white man, was first seen on April 8, 1952. He complained of a choking sensation, but had no interference with deglutition. The patient stated that at times his breathing was normal but that at other times he had some wheezing, particularly on inspiration. There was no history of hoarseness. When questioned further, he stated that there was some sensation of a foreign object in his windpipe.

The examination revealed a well-developed, well-nourished white man in no acute distress. The oto-laryngologic examination was essentially normal. Indirect laryngoscopy revealed normally appearing vocal cords. There was normal movement on phonation. During the laryngeal examination, the patient developed an inspiratory stridor. Again I examined him but could see nothing of consequence in the larynx or subglottic space. There were no masses in the neck. The thyroid was not enlarged. A roentgenogram of the chest was negative.

On April 16, 1952, a bronchoscopy was done. After preliminary medication, the airway was prepared with topical cocaine anesthesia. A 7 by 40 Jackson bronchoscope was introduced without the use of a laryngoscope. A mass was detected just beneath the right vocal cord arising from the right tracheal wall. It was smooth in outline and approximated the size of a small walnut. Measurements revealed it to be 18 cm. from the upper incisor teeth. The remainder of the tracheobronchial tree appeared to be entirely normal. Because hemorrhage

Fig. 1.—Artist's drawing illustrating tumor seen at time of operation. Numbers 1 through 5 refer to corresponding rings of trachea.



From the Charlotte Eye, Ear and Throat Hospital.

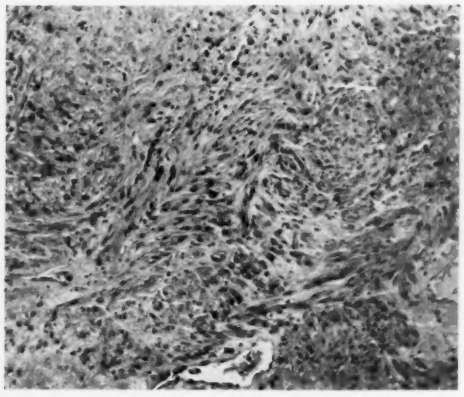


Fig. 2.—Histological elements of tissue received. The specimen is thus identified as "leiomyoma." Reduced ⅔ from mag. × 60.

from biopsy was feared, a prophylactic tracheotomy was performed with the bronchoscope left in position. The neck was prepared in the usual manner. A low tracheotomy was performed after 1% procaine infiltration. When the tracheal incision was spread, the tumor could be visualized high up on the right side of the trachea (Fig. 1). Half of the tumor was removed with a nasal snare. The remaining half of the tumor was dissected from its bed and removed, without difficulty. The base of the lesion was then electrocoagulated. A No. 5 tracheotomy tube was placed in position. The patient was returned to the room in good condition.

Decannulation was done on April 17, 1952. Except for some subcutaneous emphysema which developed postoperatively, the patient made a normal convalescence.

The report from Dr. Paul Kimmelstiel, pathologist, was as follows:

"Macroscopic: Received two formalin-fixed tissue nodules, each of which measures approximately 1.5 × 1 × 0.8 cm. The cut surfaces have a rather uniform light tan color and contain cystic spaces up to 2 mm. in greatest dimension.

"Microscopic: [Fig. 2.] Histologic examination of material received shows the tissue to be extremely cellular, the component cells being seen in both longitudinal and cross sections. As seen longitudinally, the cells are spindle-shaped elements, possessing thin spindle-shaped elements and shorter plumper nuclei. There is a moderate variation in nuclear size, shape and staining effect, but mitotic figures are not found. The spindle-shaped elements are arranged in interlacing whorls and bands. On cross section the nuclei appear round or oval and are vesicular. The cytoplasm of the cells is acidophilic and very fibrillary. Multinucleated cells, with darkly staining nuclei, are occasionally seen. Another interesting feature is the presence within some of the nuclei of a round or oval lilac-staining homogeneous body. In some areas the tissue is very loose and fibrillary, containing relatively few cells, an occasional thin-walled vessel, and small numbers of lymphocytes and plasma cells. In other areas there are seen foci of mucinous degeneration.

"In some places, between the spindle-shaped cells are found round or oval spaces which are sometimes empty and sometimes filled with an amorphous lightly acidophilic precipitate.

"Those foci exhibiting moderate disparities of nuclear size, shape and staining effect are the same foci showing areas of mucinous degeneration and other secondary degenerative changes.

"Interpretation: Leiomyoma of trachea."

SUMMARY

A case of leiomyoma of the trachea is presented. The lesion must be extremely rare, for a careful search of the literature failed to reveal any previously recorded case.

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CHEMODECTOMA OF THE LARYNX

Case Report

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and
W. H. SAUNDERS, M.D., Columbus, Ohio

CHEMODECTOMA is the name proposed by Mulligan [1] for that histologically similar group of tumors occurring in the carotid body, glomus jugulare, and certain other parts of the head and neck. It is now generally agreed that these tumors are not part of the chromaffin or paraganglionic systems, as used to be thought, but that they arise from both the mesoderm of the third branchial artery and the ectoderm of the glossopharyngeal nerve (Boyd).[2] "Chemodectoma" connotes the special function of these cells, which is to respond to chemical changes in the blood, as related both to oxygen and carbon dioxide and to certain drugs.[3] Thus the term chemodectoma is used here in preference to the older terminology, which includes both paraganglioma and nonchromaffin paraganglioma.

Chemodectomas are uncommon; not more than 350 cases have been reported, although there are many articles dealing with them. The difficulty of accurate preoperative diagnosis and the profuse and alarming hemorrhage which may complicate their surgical removal seem to assure continued interest in these neoplasms.

The classic carotid-body tumor has histologically identical counterparts which have been found in the middle ear, near the nodose ganglion of the vagus nerve, in the orbit, and in tumors associated with the aortic bodies.* In all of these locations anatomists have described normally occurring precursor cells from which chemodectomas presumably take their origin. That these same cell groups might be expected in other sites about the head and neck was emphasized by Guild [5] in his original report describing the glomus jugulare. Careful preparations from the dog and cat have shown carotid-body-like formations along several of the smaller neck vessels.[6] Israel and Bollinger [7] described a human case in which a chemodectoma was found not in the carotid bifurcation but 2.5 cm. distal to it. Other ectopic sites in the neck were reported by Monro,[8] who observed a chemodectoma in the posterior triangle, and by Rössing,[8] who reported a case with the tumor at the lower border of the thyroid cartilage and unassociated with a normal-appearing carotid body. Careful dissections in the human have shown absence of the carotid body in a small percentage of cases,[10] making one wonder if in these cases the same chemoreceptor tissue might not be present elsewhere in the neck. In the month of December, 1953, a tumor of the parotid gland removed from a 29-year-old white woman, University of Michigan Hospital (No. 771969), by Dr. J. H. Maxwell was reported on histopathological examination to be a chemodectoma.†

REPORT OF CASE

A 38-year-old Negro male prison inmate was asymptomatic until 18 months before the present illness. At that time he first noticed hoarseness, which gradually progressed to stridor and dyspnea. Four direct laryngoscopies and one laryngofissure had been performed previously in an effort to obtain a specimen from the large submucosal tumor in the left side of the larynx. None of the biopsy specimens demonstrated evidence of neoplasm, but rather "edematous, vascular tissue with chronic inflammation."

During his examination in November, 1953, the patient spoke in a muffled hoarse voice and was frankly dyspneic on exertion. He had some dysphagia for solid foods.

From the Department of Otolaryngology, University of Michigan Medical School, Ann Arbor, Mich.

* References 4 and 5.

† Maxwell, J. H.: Personal communication to the authors.

Indirect laryngoscopy revealed a submucosal mass in the left side of the larynx. The greatest prominence was in the region of the vestibule, ventricular band, and aryepiglottic fold. The true cord on the right was obscured by the mass. The entire left side was scarred and irregular, owing to previous biopsies, but not ulcerated. The pyriform recess was obliterated on the left side, and there was some pooling of secretions in the larynx. Large varicosities were present in the valleculae and aryepiglottic fold on the left. Palpation of the neck failed to reveal evidence of tumor.

The general physical examination demonstrated no abnormalities other than those described in the larynx.

Routine laboratory studies gave normal results.

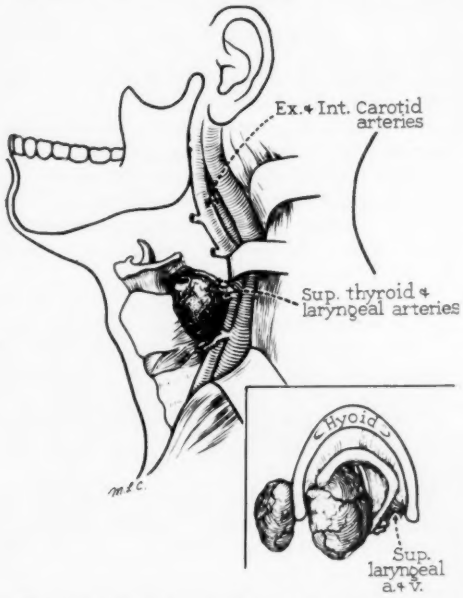

Fig. 1.—Schematic drawing showing tumor in neck and larynx.

Roentgenograms of the neck demonstrated a large soft tissue mass occupying most of the larynx. This extended upward toward the epiglottis and downward to the esophageal opening. Posteriorly it compressed the lower pharynx.

On Dec. 19, 1953, with the patient under ether anesthesia, a left lateral pharyngotomy was performed after a preliminary tracheotomy. The sternocleidomastoid muscle was retracted posteriorly and the omohyoid muscle divided. The great vessels of the neck were retracted posteriorly, exposing a remarkably vascular and friable tumor, on the surface of which were many large vascular channels, giving the entire mass a worm-like appearance. This tumor mass in the neck lay below the hypoglossal nerve, and inferiorly it was attached by

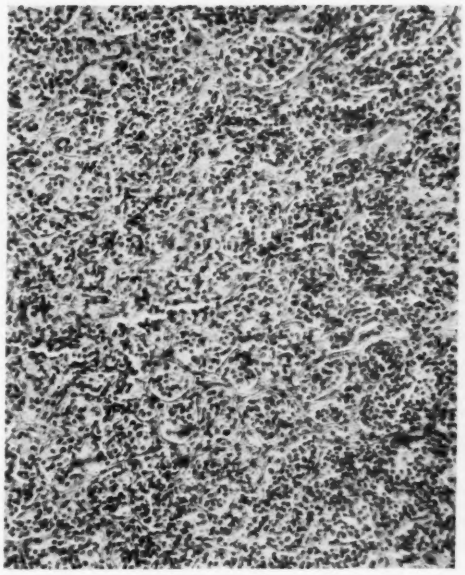

Fig. 2.—Photomicrograph of tumor, low power.

fibrous tissue to the superior pole of the thyroid gland. Laterally the tumor was adjacent to the carotid sheath but clearly not attached to it; medially the tumor passed under the ala of the thyroid cartilage and presented its greatest bulk within the larynx under cover of scarred laryngeal mucosa (Fig. 1). In removing the tumor, each large vessel along its surface was ligated. The left thyroid ala was removed, and a portion of the mucosa of

Fig. 3.—Photomicrograph of tumor, high power.

the larynx was removed with the tumor. Excision of the tumor was complete. In effect, the operation accomplished a submucous resection of the left half of the larynx. The epiglottis was left intact. Bleeding was profuse, and blood loss was estimated at 1000 cc.

The convalescence was uneventful except for several episodes of aspiration of liquids. Six months after operation there was no evidence of recurrence of the tumor and the airway was entirely adequate, although there was partial fixation of the left side of the larynx. The patient is working full time in prison, where he will remain for some time.

The tumor tissue was examined by two laboratories, and both reported chemodectoma. One report is quoted: "There is a highly vascularized stroma. Strands of fibrous tissue course from the capsule in various directions throughout the tumor. The tumor cells are polyhedral and tend to form a medullary pattern. The cells appear fairly regular in size and shape but large numbers of hyperchromatic nuclei are seen. Diagnosis is chemodectoma." A lymph node submitted showed no invasion by tumor cells.

COMMENT

It seems most probable that the chemodectoma reported here had its origin in the region of the laryngeal ventricle under cover of the ala of the thyroid cartilage, and possibly from undescribed precursor cells along the course of the superior laryngeal vessels and nerve. The greater part of the tumor grew medially and displaced the left ventricular band well past the midline. As the tumor grew still larger, it apparently spread laterally and came to lie in the neck outside of the larynx, having dissected a route along the course of the superior laryngeal vessels and nerve. The segment of tumor in the neck external to the constrictors of the pharynx was definitely smaller than that in the larynx. No part of the tumor was connected to the region of the bifurcation of the carotid artery. The tumor was completely excised, and no metastatic nodes were found in the neck. This point is worthy of emphasis in light of the recent reports by Romanski [10] and by Morfit, Swan, and Taylor,[11] which indicate clearly that chemodectomas in the neck may be locally invasive and also give rise to distant and fatal metastasis. It had previously been the general consensus that these neck tumors were benign except by position.

SUMMARY

This report is based on a case of chemodectoma of the larynx, believed to be the first of its kind recorded. The diagnosis was unsuspected, although the profuse bleeding reported at the time of preliminary biopsies might have aroused suspicion. In this case a small neck tumor found at operation was only an extension of a larger submucosal laryngeal tumor, which accounted for all the patient's symptoms. A lateral pharyngotomy was performed to resect the laryngeal tumor and its cervical extension. The patient is alive and well six months after the operation.

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Clinical Notes, New Instruments and Techniques

A PERFORATING CHISEL FOR COMPLETING NASAL FRACTURE

A New Instrument for Use in Rhinoplasty

DAVID MYERS, M.D., Philadelphia

The perforating chisel (Fig. 1) was developed as an aid to producing the nasal fracture after lateral osteotomy during the course of a complete rhinoplasty. Techniques to produce this fracture vary. Some surgeons outfracture with a chisel between the nasal bones. Others use heavy pressure with the thumb, producing the fracture. Sometimes this fracture occurs very readily, but at times great pressure is exerted and still it does not occur. At other times, the fracture line may occur in an irregular manner which one cannot control. In order to overcome this, I developed this instrument, which is used somewhat like an awl. A series of perforations are placed in the nasal bone, from the external limit of the nasal bone to the region of the inner canthus, where the lateral osteotomy ends. After the perforations (which vary from one to three) are made, it is then easy to complete the fracture, either with the chisel and outfracturing or with thumb pressure to produce a fracture along the preconceived line. The nasal bone will break

From the Department of Otorhinology, Temple University School of Medicine.

Fig. 1.—The perforating chisel, as made by the B. J. Florsheim Company.

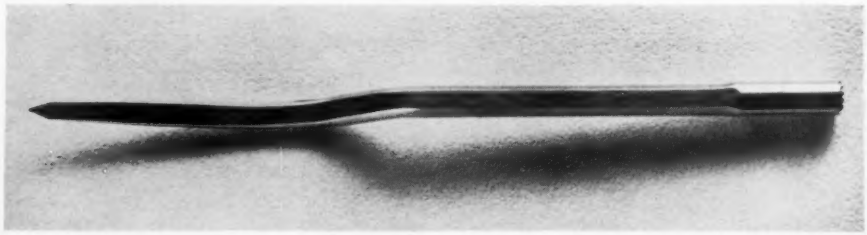

Fig. 2.—The perforating chisel is introduced under the skin to the desired point of fracture. Then is struck a sharp blow.

Fig. 3.—The actual point of entrance of the chisel is shown, to illustrate the use of the chisel more clearly. However, in actual fracture the chisel is used as in Figure 2.

along the line of perforation somewhat as one tears a postage stamp. This chisel was made for me by the B. J. Florsheim Company, and is a modification of the Berger chisel used in rhinoplastic surgery, which is made by the same company. To date, I have not seen this instrument illustrated in the literature. No great revolution of rhinoplasty technique occurs as the result of this instrument, but in my hands it has made a sometimes difficult portion of the operation somewhat easier.

The technique is very simple: After the lateral osteotomy has been produced, the chisel is introduced under the skin to the line of the desired fracture (Figs. 2 and 3). The point of the instrument is placed into the bone, and a sharp blow is struck with the mallet, actually driving the chisel through the full depth of the bone. The same procedure is carried out at a lower level until the perforations are marked off at the desired level, and in the necessary number. The fracture is then completed in the desired way.

304 S. 19th St. (3).

REMOVAL OF STRAIGHT PIN FROM LOWER LOBE BRONCHUS

Report of a Case in Which a Westinghouse Fluoroscopic Intensifier Was Used

HERBERT H. HARRIS, M.D., Houston, Texas

H. C., a girl, aged 5, was referred on April 21, 1954, having aspirated a straight pin, which had lodged in the right lower lobe of the lung. This had occurred approximately 24 hours before she was seen by me.

Upon admission to the hospital the pin was found to be lying in the anterior part of the lower right lobe, with the point upward at about the level of the diaphragm in a PA projection (Fig. *A*). In the lateral projection the pin can be seen in the anterior part of the lower lobe (Fig. *B*). About 24 hours after admission, and after all details had been carefully considered, it was concluded that this would necessarily have to be removed under fluoroscopy. The only available fluoroscope which we thought would be adequate to use in this case was the fluoroscopic intensifier (Fig. *C*), which was manufactured as its first commercial unit by the Westinghouse Company and was being used for research work at the Southwest Poliomyelitis Respiratory Center.

Because of the age of the child, it was considered best to do this under general anesthetic. Because of certain open circuits around the fluoroscopic table and intensifier, the patient was anesthetized under ether in an adjoining room and then transferred to the fluoroscopic room. A 4 mm. by 30 cm. Holinger-Jackson bronchoscope was passed into the lower

From the Department of Otolaryngology, Baylor University College of Medicine.

Fig. *A*.—PA projection shows the head of the pin slightly above the diaphragm.

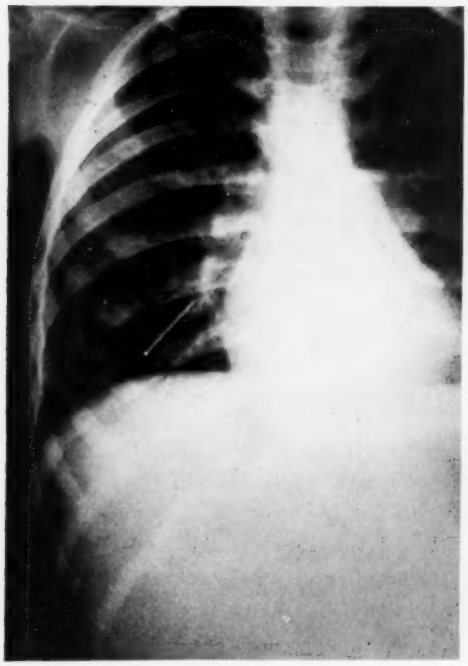

Fig. *B*.—Straight pin seen with point upward, probably located in the anterior basal bronchus of right lower lobe.

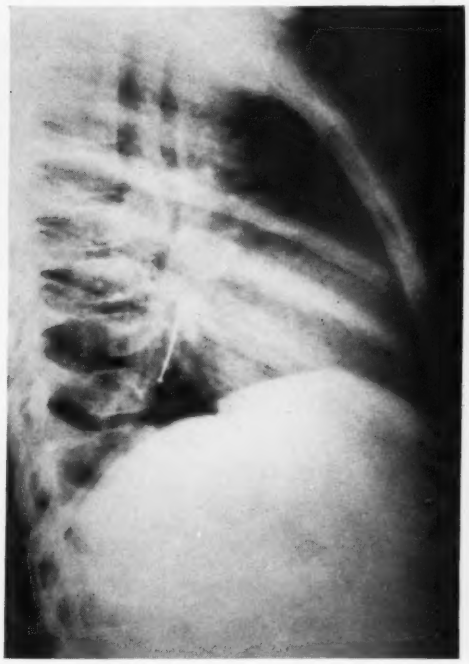

Fig. *C*.—The fluoroscopic intensifier may be attached to any fluoroscopic screen. The viewing is done in the mirror at the side and is quite satisfactorily done in a dimly lighted room.

lobe region. With the mosquito side curved forceps, probing was done under direction of the operator of the fluoroscopic intensifier. His description of the approach to this pin was quite dramatic. According to his interpretation, at no time did he feel lost and at all times did he feel that he had depth perception. In his detailed description he said that the pin was grasped at the first attempt and removed through the bronchoscope. This pin was probably in one of the tertiary bronchi, since it was about 5 cm. below the lowest point that a 4-mm. Holinger bronchoscope could be passed in a 5-year-old child.

This child recovered, without any further difficulty, having developed no pneumonitis as a result of the manipulation.

COMMENT

The detailed description given by the operator of this fluoroscope, which apparently was made possible by the clearness of the image and its relationship to surrounding structures, leads one to believe that this may be used with as much advantage as the biplane fluoroscope or the stereofluoroscope. It has been used by me in such other detailed studies as the normal and pathological conditions of swallowing.

1204 Hermann Professional Building (25).

Progress in Otolaryngology

OTITIS MEDIA AND COMPLICATIONS

Summaries of the Bibliographic Material Available in the Field of Otolaryngology for 1953

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THE SUMMARIES of the 1953 articles on otitis media and complications are listed under several headings. In a few unobtainable papers the abstracts found in *Excerpta Medica* were used. Comments of the reviewer appear in brackets.

BACTERIOLOGY

Dawes [1] (Oxford, England) reported nine cases of otogenic encephalitis in patients also having myringitis bullosa hemorrhagica. His summary of the literature is very complete. He agrees with others that the organism is probably a virus, possibly the herpes simplex. His patients all recovered, although some had very severe encephalitis. Some developed coma and epileptiform convulsions within a few hours of the appearance of the ear vesicles. Intense pain was characteristic and was not relieved with the onset of discharge, as is usual with acute otitis media. Three cases developed nerve deafness, one of whom had permanent deafness in one ear together with destruction of the labyrinthine function of the same ear. Comparison is made with the complications seen in the encephalitis sometimes seen in mumps, probably a similar virus disease. He thinks that some cases of Bell's palsy with severe pain may be caused by the virus of myringitis bullosa, even though the characteristic vesicles and bullae do not appear.

Wright and Guilford [2] (Houston, Texas) wrote about the importance of bacteriology in ear infections. They classified all ear infections as follows:

I. Infections arising by way of the Eustachian tube
II. Infections arising by way of the external canal

In the first group the organisms are usually Gram-positive and are almost invariably susceptible to penicillin or one of the newer antibiotics. Those of the second group are almost always Gram-negative and may be considered as external otitis, even though they may include cases of acute or chronic otitis media infected through a perforated drum. These Gram-negative organisms are not sensitive to penicillin. Pseudomonas was found to be the causative organism 49 times in 81 cases.

In sensitivity tests on 20 strains of Pseudomonas, polymyxin produced the highest per cent of inhibitions of growth and was found to be the drug of choice. They advised using medications for external otitis in the form of powder and found the Upjohn penicillin blower to be economical. In the acute stage of external otitis, codeine or Meperidine (Demerol) is given by mouth for relief of pain. When swelling had subsided, a powder was dusted in: chlortetracycline (Aureomycin), neomycin, or polymyxin. Careful sterility in handling cases recently operated on was urged.

Lahikainen [3] (Turku, Finland) did a very careful bacteriological study on exudate aspirated from the middle ears of patients with acute otitis media. Seven hundred thirty-four ears of 566 patients were aspirated. Children were given general anesthesia, and the adults were given local anesthesia of 10% cocaine in analine oil. The drums were cleaned with alcohol to prevent contamination of the cultures. Sensitivity tests were done on the isolated bacteria. The study covered two and one-half years. Cases in which discharge had begun spontaneously were not included in this series. He found that drainage through the puncture opening was adequate and only rarely did the opening close before the infec-

tion was healed. Repetition of the aspiration was needed in only about 5% of the cases. Penicillin was used in 176 cases; the rest of the series that could be followed through the whole illness—453 in number—received no antibiotic and no sulfonamide. There were no complications in the penicillin-treated group and only nine (2%) in the untreated group. Seven cases developed mastoiditis, one developed meningitis, and one lateral sinus thrombosis and brain abscess. This was the only fatality. The meningitis case was due to Streptococcus viridans. He was able to find only two other cases reported as developing meningitis from a viridans otitis. The fatality from lateral sinus thrombosis and brain abscess was due to the Pneumococcus Type III organism. He remarks:

> If we consider in particular the abrupt fall in the frequency of complications in Pneumococcus type III infections, keeping in mind the altered character of this disease previously so dreaded, it is difficult to find any other explanation than that the virulence of the bacterium has changed in the course of years.

There were 81 cases from which Pneumococcus Type III was cultured, 201 cases in which other types of pneumococci were found, 177 were S. pyogenes, 24 S. viridans, 112 H. influenzae, and 126 in which there was no growth. Results of the sensitization tests were interesting. All of the H. influenzae were sensitive to sulfathiazole and none to penicillin. No other organisms were resistant to penicillin. He noted also that there was a very definite bacteriostatic effect produced by fluid exudate in cases infected by streptococci and pneumococci, but not in cases infected with H. influenzae. [A very interesting paper.]

CHEMOTHERAPY AND BIOTHERAPY

Welch [4] (Director of Antibiotics, Food and Drug Administration, Washington, D. C.) edited this year the first Antibiotics Annual, a symposium on antibiotics held at Washington, D. C., in October, 1953. The Antibiotics Annual is a book of 632 pages containing 105 articles, most of them reports of laboratory workers concerning many new antibiotics, such as hygromycin, streptogramin, ruticin, streptocardin, methymycin, puromycin, and nystatin. Several of the reports are written by clinicians and give comparative results of different antibiotics. A few of these articles have been abstracted and appear in this review.

Perrin Long [5] (New York) summarized reports of 19 anaphylactic reactions to penicillin, 11 of which were fatal. He commented that a number of fatal reactions occurred in patients that had had definite reactions to penicillin on previous occasions. He therefore advised careful questioning of each patient as to previous reactions. Patients with allergic tendencies, "as for example, those ill with bronchial asthma—should not be treated with penicillin for fear of inducing a state of anaphylaxis." Procaine reactions are not uncommon and penicillin procaine may produce fatal reactions in patients who are sensitive to procaine. He warned that serious reactions are not rare and that most reactions are never reported.

Schenck [6] (Philadelphia), in discussing the use of antibiotics without paracentesis in otitis media, said:

> When, in spite of antibiotic therapy, frank bulging of the eardrum persists for more than 24 hours, paracentesis is imperative if serious damage to the conduction apparatus is to be avoided.

He also advised audiograms after every attack of otitis media in order that additional treatment may clear up as much hearing loss as possible.

Rutherford [7] (Oakland, Calif.) reported 150 cases of acute otitis media seen in private practice in two years. Only cases severe enough to require incision of the drum were included. Of these, 24% had already undergone spontaneous rupture. Only 28% of the group were given any antibiotic. All recovered without mastoidectomy with dry ears; duration of discharge was 4 to 14 days. She strongly advises more careful observation of each case and does not use the antibacterials unless recovery is slow or complications threaten.

In the discussion Bigler (a pediatrician) said that the experience at the Children's Memorial Hospital (Chicago) was quite the opposite.

> Since the general use of antimicrobials, our incidence of mastoidectomy has decreased from 114 in 1940 to 1 in 1950. . . . Our incidence of myringotomies has followed about the same pattern. . . . I cannot go along with the school of thought that states that the antimicrobials should be used only after conservative methods of therapy have proved inadequate . . . failures are due to late therapy and discontinuance too soon.

McLaurin [8] (Baton Rouge, La.) urges the use of myringotomy in acute otitis media and does not use antibiotics unless temperature remains high, the patient remains toxic, or unless improvement is not noticed within 24 hours after myringotomy. He believes:

> When otitis media is incompletely resolved, as it frequently is when it is inadequately treated with even the most potent of the new agents, adhesions form in the ossicular chain; and the regional mucosa becomes thickened and fibrotic.

In one month he treated 55 cases of otitis media. Thirty-four cases had no previous treatment, and all recovered with myringotomy in an average of 5 to 12 days. The remaining 21 cases had been given antibacterial medications without myringotomy. Myringotomy was done on these 21 cases, but their recovery required an average of 13 to 55 days and 9 required antibiotics.

Nákó and Hajts [9] (Budapest, Hungary) reported a series of 40 cases of acute mastoiditis that did not heal with inadequate penicillin therapy, all requiring simple mastoidectomy. They observed that these cases were apt to progress slowly, with few or no symptoms. There would frequently be a violent exacerbation of symptoms several weeks after the infection was considered to be cured. They suggested more prolonged penicillin therapy and checks by x-ray and sedimentation tests.

Breese [10] (Rochester, N. Y.) reported on 1204 beta hemolytic streptococcic infections seen in a pediatric practice during five years, and compared methods of treatment. These infections included 52 cases of sinusitis and acute otitis media. He is at present using a single intramuscular injection of 600,000 units of penicillin G benzathine (Bicillin), which is reported to give an effective blood level for two weeks. Using this treatment, he found no recurrences of infections in the first two weeks and only 5% recurrences in one month.

Bunn [11] (Syracuse, N. Y.) reviewed the use of the newer antibacterial agents and emphasized the point that whatever the medication used in severe infections, it should be continued at least five days. He used penicillin almost altogether by mouth. He stated that "by all odds penicillin is the best drug for Gram-positive bacteria."

Heller [12] (Englewood, N. J.) reported a series of 1031 cases of acute otitis media seen in a private pediatric practice during the past six years. No mastoid operations were necessary, and myringotomy was done in only 1.3% of the cases. The great majority of cases cleared up in two days. The usual treatment was penicillin intramuscularly for one dose followed by penicillin by mouth, or sulfonamides alone or in combination with penicillin. He states:

> I believe that these results indicate that myringotomy is, in most cases, an unnecessary procedure. . . . In older, co-operative children I avoid injection, both from the standpoint of needless pain and the lesser likelihood of reaction when the substance is given orally. . . . I ask that the oral medication be continued at least 4 days.

Wheatley [13] (England) reported on the results of 93 cases of acute otitis media. Of these, 61 were treated with oral penicillin or oral penicillin and a sulfonamide. Nineteen had perforated before treatment was begun. No complications occurred, no mastoidectomy operations were necessary, and all regained normal hearing, with no permanent perforations. He was convinced that the oral administration of penicillin was effective and that the addition of a sulfonamide increased its efficiency. He avoided early incision of the drum, as he felt that pus, even though present, would drain away later through the Eustachian tube without formation of adhesions, with later hearing loss. Most of his cases were observed in private practice and

were seen for other conditions as late as two or three years afterward, so he was convinced that there was no resulting hearing loss.

Zonderman [14] (Boston) reported two cases of otitis media that had been treated with inadequate doses of penicillin. One case requiring mastoidectomy was due to Pseudomonas organisms that were insensitive to penicillin and most of the broad-spectrum antibiotics. He urged that cases of otitis media be followed more carefully and that adequate doses of antibiotics be given for a longer time. He also vehemently condemned the use of antibiotic lozenges, "which are not only useless but have produced a host of complications, including angioneurotic edema and severe stomatitis."

[These papers show the difference of opinion existing concerning the necessity for early myringotomy to prevent adhesions and resulting loss of hearing. Certain infections, especially those caused by the Pneumococcus Type III, are prone to cause destruction of tissue and adhesions, even in apparently mild infections. We used to see, before penicillin, an eardrum melt away in a few days in cases of scarlet fever, in spite of early, wide incision. Certainly the majority of cases of otitis media do not develop adhesions, whether incised early or not at all. But in those cases that do not respond in a day or two to penicillin, incision should be done and the patient followed carefully until hearing becomes normal.]

CHRONIC ADHESIVE OTITIS

Ojala [15] (Finland) reported the histological study of 116 temporal bones which were examined for adhesive otitis. Seventeen of the temporal bones (representing 12 patients) had severe adhesions, and 30 bones (26 patients) had moderate adhesions. Almost all the severe cases showed associated healed mastoiditis, and many with mild adhesions showed mastoid damage. All these persons were aged, over 70 years average, therefore it was not possible to correlate hearing loss as observed before death. He quoted Haymann's animal experiments to show that in infections produced by the Pneumococcus Type III adhesions begin in the first three or four days of inflammation. With other organisms, such as in diphtheritic otitis, adhesions rarely occur. He concluded that early drainage of acute otitis media and serous otitis should help to prevent later deafness. His illustrations are excellent.

CHRONIC OTITIS MEDIA

Versteegh [16] (Holland) reported two cases in which a marked hearing loss followed explosion trauma. Within six months both cases developed symptoms of attic infection, with occasional slight drainage and pain. One case was found to have the incus moderately eroded by infection, and in the second case the incus could not be found. He assumed that the force of the explosion had partially or completely dislocated the incus, destroyed its precarious blood supply, and started anemic necrosis of the ossicle. Although he did not see either case directly after the accident, the history did not suggest a rupture of the drum by the explosion.

He also reported that in five cases in which conservative radical mastoidectomy operation had disclosed a normal incus and other ossicles the improvement in hearing following operation had averaged 17 db. In 13 other cases the incus was so far necrosed as to require removal. The improvement in hearing after operation averaged only 9 db. He therefore urged early modified radical operations in order to conserve hearing.

Moore [17] (New York) presented 18 cases of chronic otitis media in which endaural modified mastoidectomies had been done. The average duration of drainage before operation was 13 years. His results were excellent. Dry cavities were obtained in 83% in an average time of 10 weeks. Hearing was improved in seven cases, no change in five, and some additional loss in five. (One case did not have a follow-up audiogram.)

The decision between radical and modified procedures was decided at the operating table after inspection (with magnification) of the attic and tympanic areas. He believes in re-

taining the matrix of the cholesteatoma in cases in which the middle ear had not been invaded.

In the discussion Williams and Walsh disagreed with the last statement and advised complete removal of the matrix. Day stated that "the preservation of even a crescent sliver of the drum is important" in the effort to conserve hearing.

Tionloc [18] (Manila) tried cortisone drops in a series of 14 cases of chronic otitis media. Three cases cleared up; the others responded to penicillin and dihydrostreptomycin given locally and parenterally. Even in those cases in which further treatment was necessary, the granulations became less red and swollen and drainage was diminished. [Since cortisone may inhibit defense reactions in infections, it should be used with great care in such cases.]

Peele [19] (Kinston, N. C.) reported on the local use of nitrofurazone (Furacin) in the treatment of 55 patients with chronic otitis media. Most of these cases had been treated by other methods, with no improvement. He obtained dry ears in 78% in the series. He observed sensitivity reactions in 12.7%.

In cases with persistent perforations of the drum he was able to obtain healing in 59.7% by repeated applications of 50% trichloroacetic acid to the edges of the perforation. An average of 14 treatments were necessary. Applications of the acid were made at weekly intervals.

McGuckin, Livingstone, and James [20] (England) each discussed the management of chronic otitis media at the meeting of the Royal Society of Medicine, Section of Otology. Their discussions were very interesting and well written. McGuckin stated that "We are now seeing an increasing proportion of cases in which the disease is already chronic when the patient first has a symptom of any kind." He emphasized that operation for chronic otitis media should not be primarily for drainage but for removal of all traces of cholesteatoma matrix, diseased bone, and granulation tissue.

In the discussion, Tumarkin said he had examined 100 children from a poor district and found 73% of them to have abnormal drums, 5% to have dry perforations, and 1.5% to have frank otorrhea.

CHOLESTEATOMA

In a symposium on surgical management of aural cholesteatoma at the Academy of Ophthalmology and Otolaryngology, Walsh presented his reasons for removing the matrix during operations for cholesteatoma, Baron told "Why and When I Do Not Remove the Matrix," and Lindsay spoke on the pathogenesis of cholesteatoma and summarized the discussion on removal of the matrix.

Walsh [21] (St. Louis) reported a case in which, following radical mastoidectomy, a fistula developed much later into the labyrinth beneath the matrix. He suspected that underneath the retained matrix there may be absorption of bone, and thinks that this may happen because of a chemical osteolytic process.

Baron [22] (San Francisco) leaves in the matrix, when he considers it safe, in order to aid the epithelization of the cavity and to conserve hearing. He thinks that the matrix should be removed when granulations or infections are under the matrix, and is suspicious of infection when the matrix has a pinkish color. When the mastoid is highly pneumatized the matrix should be removed, where necessary, to remove cells. He quoted Shambaugh, who stated, "I fail to see any difference between stratified squamous epithelium that lines a cholesteatoma and that lining a healed radical cavity."

Lindsay [23] (Chicago), in discussing the pathogenesis of cholesteatoma, stated that the mass of evidence, clinical and histopathological, indicates an origin from the epidermis of the canal or drum membrane. The metaplasia theory lacks the support of convincing histopathological evidence.

He divided cholesteatomas into two groups. The first group consists of those with extension of the epidermis through a marginal perforation. This group has become more

rare since the use of antibiotics and the resulting decrease of necrotizing otitis media. The second group has shown no definite decrease in numbers and probably arises as a result of repeated middle-ear infections and tubal obstructions. In such cases the air spaces in the attic are filled by proliferated fibrous connective tissue, which probably contracts, causing retraction of Shrapnell's membrane. With each inflammatory episode the epidermis is stimulated to activity, growth, and rapid exfoliation. Retention of exfoliated cells and crusted exudate in the retracted pocket may occur. The absence of a fibrous middle layer in Shrapnell's membrane probably favors retraction as well as extension medially of the actively proliferating epidermis into the connective tissue of the attic. Retention of desquamated cells and crusted exudate during an ensuing acute inflammatory episode provides the nucleus of the cholesteatoma.

In summarizing the papers of Baron and Walsh, he stated that his clinical experience agreed with Baron that there was no evidence that a smooth matrix would cause recurrences unless there remained chronic infection, moisture, and crusting.

Meltzer [24] (Boston) reported 43 cases of cholesteatosis in children, most of which were unrecognized until late in the course of the disease. These cases were found in the records of the Massachusetts Eye and Ear Infirmary in the 15-year period of 1937 to 1952. The children were all under the age of 10 years. (There were 464 similar cases above the age of 10 years.) He was impressed that the diagnosis was not made early enough to save hearing and that too much time was wasted by "such temporizing treatment as repeated adenoidectomies, removals of polyps or granulations, and simple mastoidectomies. . . . In the typical case the only sign of trouble is discharge—almost always malodorous. Sometimes there is not even a discharge." Many cases showed no bone destruction by x-ray. He strongly advised early modified radical mastoidectomy to preserve hearing. He believes that cholesteatoma may be due to metaplasia (a desquamative inflammatory reaction set up in the epithelium of the middle ear) or ingrowth of epithelium through a marginal perforation.

Harpman [25] (Warwick, England) presented in some detail eight cases of cholesteatoma. One 24-year-old patient had bilateral cholesteatoma; one ear had an intact drum and no history of any drainage. His second case, a boy of 14, had a large cholesteatoma in an ear that had drained for only 10 days. Examination had showed a normal drum some months before. He concluded that cholesteatoma may be of congenital origin in some cases.

House [26] (Los Angeles) reported the case of a 26-year-old woman with an apparent primary cholesteatoma. The patient had tinnitus and fullness for six months and severe vertigo for a few weeks. X-rays and neurological examinations were negative, but operation revealed a small cholesteatoma, only 4 mm. in diameter, occupying the region of the stapes. Radical mastoidectomy was done, and there have been no more symptoms.

Hughes and McKenzie [27] (London) discussed the problem of when cholesteatoma cases should be operated upon. They state that in the past they hesitated to operate on patients with cholesteatoma because of the impairment of hearing which frequently followed. Recently they examined 50 cases chosen at random from a large number on which modified radicals had been done. None of these cases showed hearing loss greater after operation than before.

Out of 41 cases recently operated on for chronic otorrhea, 28 were found to have cholesteatoma. They think that this frequency of cholesteatoma supports the theory of congenital origin of cholesteatoma.

They also remarked that bone conduction is remarkably good in cholesteatoma, no matter how much hearing had been lost by air conduction.

Juers [29] (Louisville) agrees with Day that cholesteatoma is most frequently due to the attic-retraction type of perforation. The first step is the sealing-off of the attic cavity from inflammatory reactions. His diagrams are

very clear. He believes in retaining the matrix where the middle ear has remained sealed off from the attic, but that any matrix extending toward the mastoid tip should be removed to prevent formation of cholesteatomatous cysts. In five of his modified radical cases the incus had been destroyed or was removed at operation, yet their hearing level remained at a 20-db. average; therefore, the surgeon should not hesitate to remove the incus and head of the malleus where there is necrosis but should save as much of the drum and middle ear as possible.

MENINGITIS

Maxwell [30] (Ann Arbor, Mich.) reported two cases of recurring meningitis. The first case was that of a boy of 5 who suffered a skull fracture and nine days later developed his first attack of meningitis. A second attack occurred five months later. The third attack occurred 10 months after the accident, and a fourth attack occurred a year later. This time x-rays revealed a fracture line in the petrous pyramid, which previous x-rays had failed to demonstrate. Operation was done with the help of neurosurgeons, and a trapping of dura was found in the fracture line. The dura was freed, elevated, and a strip of Gelfoam was placed over the fracture line. The patient has remained well for three years. The second case resulted from a spread of otitis media to the stalk of a meningocele which penetrated a pneumatic cell in the petrous apex. Separation of the stalk and packing with absorbable gelatin sponge (Gelfoam) have prevented recurrences. There had been three attacks of meningitis. [Two very instructive cases.]

Richter [31] (Germany) reported a case of recurring meningitis in an infant of 7 months. In spite of large doses of penicillin, there were three recurrences of meningitis in a period of two months. During the fourth attack mastoidectomy was done. The antrum contained foul pus, but no extradural abscess was found. The infant remained well after the mastoidectomy.

LATERAL SINUS THROMBOSIS

The staff [32] of the Children's Hospital, of Los Angeles, reported an interesting case of lateral sinus thrombosis. Bilateral mastoiditis developed after an untreated case of scarlet fever. The child, 6 years of age, had an overwhelming septicemia, a pulse of 220, and was almost moribund when admitted to the hospital. Transfusions were started, 1,000,000 units of penicillin were given at once, and 2 gm. of oxytetracycline (Terramycin) given daily. Blood cultures were positive for beta hemolytic Streptococcus, and the same organisms were found in the discharge from both ears. Both drums were found to be completely destroyed. Mastoidectomy revealed complete necrosis of all cells, an extradural abscess was found, and there was thrombosis of one lateral sinus. Recovery followed without ligation of the lateral sinus. [Such cases, once so common, are fortunately rare now.]

Bradburn [33] (Phoenix, Ariz.) reported the unusual case of a 4-year-old Navajo girl with gonorrheal mastoiditis and petrositis, tuberculous mastoiditis and petrositis, gonorrheal vulvovaginitis, generalized tuberculosis, and lateral sinus thrombosis.

Operation revealed a completely caseous mastoid cavity and lateral sinus thrombosis. Treatment with penicillin and streptomycin improved the patient's condition, but there was a continued low-grade fever until the patient was taken from the hospital against advice on the 54th day. On the third day after mastoidectomy, and one day before the lateral sinus was opened and packed off, the temperature ranged from 94 to 106 F., a fluctuation of 12 degrees. This fluctuation occurred even when she was getting large doses of penicillin and streptomycin. Three days after the lateral sinus was packed the violent fluctuations stopped. Excision of an axillary lymph node showed tuberculosis. It was also interesting to note that a diagnostic puncture of the lateral sinus produced a free flow of blood but on opening the sinus there was an infected mural thrombus.

Cawthorne [34] (London), in a Hunterian Lecture before the Royal College of Surgeons

of England, gave a very readable summary of his views on surgery of the temporal bone. The greatest change in present and past problems is shown by the list of temporal bone operations done at King's College Hospital in 1934 and in 1951. In 1934, 98% of temporal bone operations were for relief of suppurative conditions. In 1951 only 25% were for suppuration. He stated that lateral sinus thrombosis could usually now be cared for without opening the sinus or ligation of the jugular vein. He described such a case recently seen in which a man of 25 had a swinging temperature and symptoms of meningitis secondary to a recent acute otitis media. At operation, the lateral sinus was found to be thrombosed and a large collection of extradural pus was found in the posterior fossa. After exenteration of the mastoid cells and evacuation of the pus, the mastoid incision was sutured without drainage and without opening the lateral sinus.

Penicillin was continued as before and the temperature fell to normal within 12 hours and never rose above normal again. The hearing was normal in a week, and the patient was back at work within a month.

BRAIN ABSCESS

Weiss [35] (Berlin) reported the unusual case of a boy that at the age of 9 developed a temporal lobe brain abscess secondary to a cholesteatoma. The abscess was opened, and a radical mastoidectomy was done. A persistent prolapse of the brain formed in the ear cavity. After 19½ years an abscess developed in the prolapsed portion following an upper respiratory infection. The abscess was opened, but meningitis recurred twice. Sulfonamides and repeated lumbar punctures resulted in a cure.

Sultana [36] (London) reported the case of an infant 8 weeks old who develpoed both an extradural abscess and a subperiosteal abscess as complications of an otitis media of two weeks. The child recovered rapidly after simple mastoidectomy.

OTITIC HYDROCEPHALUS

McAlpine [37] (London) reported a case of "toxic hydrocephalus" in a girl of 16. The onset followed a streptococcal tonsillitis, which responded to sulfonamides. Two weeks later a sixth nerve palsy developed, which was followed by papilledema. Spinal tap showed a pressure of 290 mm. There was no infection of the middle ears or sinuses. The infected tonsils were removed, and the increased intracranial pressure quickly cleared up. It is postulated that there is an overproduction of cerebrospinal fluid as a result of sensitization of capillaries of the choroid plexus. The thrombotic form of hydrocephalus results from failure of absorption of the cerebrospinal fluid as a result of thrombosis of the superior longitudinal sinus. This is usually due to lateral sinus thrombosis from otitis media. [This abstract was obtained from *Excerpta Medica.*]

Tamari and Henner [38] (Chicago) reported three interesting cases of intracranial complications all developing in spite of prolonged penicillin therapy. Their second case, a boy of 6, developed otitic hydrocephalus with such great intracranial pressure he was left permanently blind in both eyes. The hydrocephalus was secondary to lateral sinus thrombosis and probably extensions to other sinuses. Since daily spinal punctures did not lower the intracranial pressure, he was transferred to the neurosurgical service for constant ventricular drainage. Exploration found no brain abscess. At one time the pressure recorded during spinal tap was 480 mm.

[Otitic hydrocephalus has not been reported often in the United States. There have been a number of reports from England of this condition, based chiefly on the work of Symonds. The pressure must be kept down by frequent spinal taps. Most cases recover slowly, without permanent damage. The above case was probably damaged before admission to the hospital.]

Martin [39] (Liverpool, England) reported a case of otitic hydrocephalus occurring in a man of 32. He had had a draining ear for 11 years, without pain or headache. On admission his temperature was 104 F, and he was chilling. Operation showed the lateral sinus to be necrotic. Pus and clot were removed

backward until free bleeding was obtained. Since free bleeding could not be obtained below, the internal jugular vein was ligated. When sixth nerve palsy and severe frontal headache developed 17 days later, the simple mastoidectomy was converted to a radical and all remaining cells opened up. Papilledema was noted on the fourth postoperative day and three weeks later increased to five diopters. Repeated lumbar punctures showed great increase in pressure, over 300 mm., but the spinal fluid remained clear. Neurological consultation agreed with the diagnosis of otitic hydrocephalus, although preferring the term "cortical thrombophlebitis." Papilledema gradually disappeared in six months, and the patient remained well except for a slight sixth nerve palsy. Martin agreed with Symonds that the mural thrombosis had probably spread up into the superior longitudinal sinus, with interference of absorption of cerebrospinal fluid, thereby causing the increase in intracranial pressure. He intimated that recovery might have been faster if the lateral sinus had not been destroyed.

SEROUS OTITIS

Harcourt and Brown [40] (Washington, D. C.) observed 39 cases of secretory otitis media and suggested the name "hydrotympanum." These cases were seen at the Walter Reed Army Medical Center in patients from all parts of the world "in whom the disease developed without respect to climate, race or age." Their treatment consisted of aspiration of fluid with a 22-gauge spinal needle, which had been bent slightly so the tip could be seen past a 5-cc. syringe. In cases in which the fluid was too thick, the drum was incised and the tube inflated. No eosinophile cells were found. Several children cleared up quickly after removal of adenoids. They advise the Papanicolaou stain to rule out tumors, since in 7 of their 12 cases of malignancy of the nasopharynx there was hydrotympanum present as a very early complaint, frequently years before the diagnosis was made.

King [41] (Atlanta) presented 56 cases of fluid in the middle ear of children under 10 years of age. In each case adenoids (and usually tonsils) were removed, myringotomy was done, and the fluid was examined for eosinophiles and bacteria. In 11 cases bacteria were found, which were considered as evidence of low-grade infection. Clinical evidence also indicated infection in these cases. Nine cases showed eosinophiles in thick secretion. Cases having thin fluid showed no eosinophiles. These cases had additional symptoms of allergy and were found to be more slow in their recovery. Chlortetracycline was found to help recovery more than penicillin in cases in which infection seemed to be present. All cases recovered except three that were still having intermittent symptoms several months after operation.

TUBERCULOUS OTITIS MEDIA

Wallner [42] (Chicago) reported on 52 patients with tuberculous otitis media seen in the past five years as consultant to two large tuberculosis hospitals. During the same time 200 patients had the diagnosis of tuberculous laryngitis. Cases seen early, before perforation, were usually controlled by systemic streptomycin and perforation prevented. He approved Myerson's statement that "a discharge from the middle ear appearing without pain in a tuberculous individual should be considered tuberculous." There are many excellent photographs in the article. No operations in the mastoid were necessary; all cases responded well to streptomycin or dihydrostreptomycin.

EXTERNAL OTITIS

Leshin [43] (Chicago) studied the bacteriology of 620 cases of external otitis seen while he was stationed in the Solomon Islands, and tried to correlate the clinical pictures seen with the organisms found. He agrees with others that hot humid weather greatly increases the incidence of otitis externa. He found fungous infections frequently and described their clinical picture as follows: The

moist type is commoner, and the ear is apt to be filled with masses of material resembling wet paper. Pain is slight. The bacterial type may be very severe, with great swelling and pain in the early stages. If not treated, these cases frequently become chronic and tended to relapse. Pseudomonas was found in 80% in the bacterial group, and 30% of the group was clinically diagnosed as fungous type. They found fungi, the Aspergillus and the Candida (Monilia) frequently, and decided that they must play some part in these infections. They were not able to diagnose the etiological organisms by the clinical appearance except in the classic cases. Therapy was not reported.

Branca [44] (Fort Pierce, Florida) thinks that external otitis may be due to a neurogenic atrophy of the ceruminous glands. The absence of wax brings in the secondary invaders: the molds and the bacteria. He finds a fairly high per cent of his cases infected by molds. He sees many more cases in Florida than when he practiced in New York. There are many more cases in the warm humid months. He commented on the many cases that look clean but have intolerable itching. He comments, as have others, that the appearance of brown wax indicates a healed ear and advises that any wax be carefully left in place. He gave detailed histories of 15 cases, some of which were prolonged in spite of varied treatment.

DIAGNOSIS

Bryan [45] (St. Louis), during a study of the cells in discharges from middle ears, found "giant phagocytes" not previously reported in aural exudates. These cells were first found in material taken during operations for acute coalescent mastoiditis. They were not found in quiescent mastoiditis. After examination of secretions in 277 cases they decided the presence of these giant phagocytes was an indication of relatively severe acute otitis media and usually mastoiditis. Absence of these giant cells indicated a relatively mild illness of short duration.

TECHNIQUE

Reading [46] (London), in speaking on the management of soft tissues in mastoidectomy, advised that at the end of a simple mastoidectomy the wound be closed without drainage. He found this to be safe if penicillin was used for the first postoperative week. He found that sometimes in young children following a simple mastoidectomy there might be re-formation of mastoid cells so completely as to suggest a very incomplete first removal. He had seen a number of cases in which the meatus became stenosed after endaural mastoidectomy. To combat this he suggested interposition of a flap of skin on at least one surface of the upper part of the skin incision. He then undercuts the skin of the posterior margin of the endaural incision, removes a little of the underlying cartilage so the skin will cover the raw posterior edge and inhibit stenosis.

Derlacki [47] (Chicago) treated 143 central perforations of the tympanic membrane and was able to get 70% of closures, even in cases with perforations as large as 7 by 8 mm. There was an average gain of 16 db. in hearing, and some patients noticed relief from tinnitus. An average of 14.6 treatments per patient was done. His technique consisted in touching the edges of the perforation with a saturated solution of trichloroacetic acid. Then the entire drum membrane was covered with a thin cotton pledget moistened with a proprietary mixture of eucalyptol, methyl salicylate, menthol, thymol, boric acid, and benzoic acid in 20% alcohol (Euthymol). The patient kept the cotton moist by applying 2 or 3 drops of Euthymol twice daily. The patient was seen and treatment repeated at weekly intervals, occasionally at intervals of two or three weeks.

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Abstracts from Current Literature

Larynx

ACUTE STENOSING LARYNGOTRACHEOBRONCHITIS. J. FALBE-HANSEN, Nord. med. **50**:1091 (Aug. 6) 1953.

The author reports on 24 cases of acute stenosing laryngotracheobronchitis in children 2 months to 7 years of age (18 boys, 6 girls). Tracheotomy was performed in all cases. Sometimes the laryngitis was subglottic without a descending infection, sometimes supraglottic without or with a descending infection. The worst prognosis was in the subglottic laryngitis with descending infection. The mortality rate was 37%. Early tracheotomy performed under general anesthesia after intubation and tracheobronchial suction is recommended.

WALLGREN, Stockholm [A. M. A. AM. J. DIS. CHILD.].

A BACTERIOLOGICAL STUDY OF ACUTE LARYNGITIS IN CHILDREN. M. BJØRNEBOE, ESTHER FRANTZEN, H. POULSEN, and KIRSTEIN ROSENDAL, Nord. med. **50**:1094 (Aug. 6) 1953.

The authors present a bacteriological study in 39 children with primary acute laryngitis. Laryngeal swabs were taken in the course of direct laryngoscopy. Pneumococci, in 8 patients; Hemophilus influenzae, 20; γ-streptococci, 37; Staphylococcus aureus, 14, and Gram-negative diplococci, 11, were among the organisms found.

WALLGREN, Stockholm [A. M. A. AM. J. DIS. CHILD.].

Nose

DISCUSSION ON THE TREATMENT OF FRONTAL SINUSITIS (ABRIDGED). MAXWELL ELLIS and OTHERS, Proc. Roy. Soc. Med. **47**:839 (Oct.) 1954.

The meeting was held on March 5, 1954, President C. P. Wilson, C.V.O., F.R.C.S., in the chair. Mr. Ellis said that frontal sinusitis is less common and less serious than formerly. Special factors are contiguous structures, vascular connexions, and the drainage mechanism. One must distinguish clearly between acute and chronic frontal infections. Treatment of the acute is conservative, with antibiotics, the sulfonamides, shrinkage of swollen membranes, and, occasionally, the making of a small opening in the floor of the sinus, but avoiding the nasofrontal duct. These measures usually suffice, but complications of osteomyelitis and periostitis may supervene. Surgery is indicated only for drainage and removal of dead tissue. The chronic type calls for surgery and the elimination of persistent pathology, such as polypi and thickened membrane. Unfortunately, any new opening to improve drainage is likely to stenose. Mr. Ellis removes the floor of the frontal, all of the anterior ethmoidal cells, and part of the pyriform aperture. Thus one establishes a short, fairly straight channel, which may be kept open by a "Portex tube," which may be kept in for as long as two years. Mr. H. D. Fairman spoke chiefly on the surgery of frontal sinusitis. He aims to destroy the infection by middle turbinectomy, resection of the septum, and the persistent use of antibiotics, as indicated. A polythene tube is set in place, and through this irrigations of the frontal are carried out three or more times daily. In persistent cases Fairman does the "Woods' Obliterative Operation," the described technique of which seems not to vary much from the American procedures. At the end of the operation he fills the cavity with penicillin-sulfathiazole powder and applies a pressure bandage. These antibiotics are continued topically for at least three weeks. He cites 11 cases in which there were good results and no complications. Mr. R. G. Macbeth then described his osteoplastic flap procedure, based on that of Gibson and Walker. (A full account of this appeared in *J. Laryng. & Otol.* **68**:465, 1954.) Mr. C. Gill-Carey gave a tabulation of results from the Massachusetts Eye and Ear Infirmary in the 15 years preceding 1944. The table concerns 276 patients, with 95 failures and a mortality of 15%. Mr. Gill-Carey thinks that most failures were due to incom-

plete removal of all diseased tissue or to stenosis of the nasal channel. Mr. R. R. Woods favors the obliterative operation but feels that the ethmoid is practically always involved and therefore must be eliminated also. Mr. V. E. Negus, who is so well known in the U. S., said that he had written his views completely in Sir St. Clair Thomson's book (1948, Ed. 5). He avoids cutting the supraorbital nerve because of subsequent neuralgia. The antrum, which is a receptacle for pus from above, should always be irrigated or, perhaps, receive surgery. Other speakers were Prof. G. Dohlman (Lund), Mr. J. C. Hogg, Mr. W. O. Lodge, and Mr. J. H. Otty. Mr. S. W. G. Hargrove believes that the primary source of frontal infection is in the ethmoid. Two types are recognized: one in the mucosa only, the other in the bone. He cannot understand how Mr. Woods can obliterate the frontal cavity, and assumes that a blood clot method is used.

ABSTRACTOR'S COMMENT: This question is always present. Obliteration seems impossible, for some space must remain, no matter what the technique used. Having had occasion to take a second look, due to recurrence in some patients, I have never seen total obliteration, and there has always been some slight discharge at least.

I. W. VOORHEES, New York.

MUCOCELE OF THE ANTERIOR ETHMOIDAL SINUSES. C. WILD and G. KLOTZ, Rev. laryng. **75**:663 (Sept.-Oct.) 1954.

The authors present two cases of mucocele of the anterior ethmoid sinuses. After a discussion of the etiopathology of these mucoceles, they state that, in general, surgical treatment intranasally is sufficient in most of the instances, particularly in those in which the mucocele involves the middle turbinates or the bulla. On the other hand, where orbital pathology has developed with exophthalmus and diplopia, the mucocele probably involves the uncinate or the paralacrimal area. Under these conditions the external approach is necessary.

PERSKY, Philadelphia.

Miscellaneous

PRESENT STATUS OF CHEMOTHERAPY IN CANCER. CORNELIUS P. RHOADS, New York J. Med. **54**:2557 (Sept. 15) 1954.

In this excellent study and report, Rhoads has brought to date the known facts concerning treatment of cancer by destructive agents produced within the body or introduced from without. He clearly states that many investigators have been working against wind and tide, without purposeful ends. Old procedures which have been found to be of doubtful value are still being reworked. Second, many have been awaiting some new concept in biology; therefore, much time and effort and money have been used to extend our knowledge of the natural sciences. After 10 years of educating the public and the profession in early diagnosis, we may draw two conclusions: First, malignancy can be detected at a stage when curative therapy is possible, and so there has been a seemingly greater number of cancer patients. Second, extirpative efforts (surgery, chiefly) have brought an improved rate of cure. The expense has been tremendous, but the lessened death rate in certain neoplasms accessible to present usable agents has been considerable. However, there has been a greatly increased death rate in general because of the greater age of our population, and, most of all, there has been an alarming increase in discovered cancer of the lung. The situation is not as yet satisfactory, and, obviously, we must prepare other plans of action. Something new is required. It is probable that some 65% of all patients with cancer will fall into the "disseminated group," which will increase in numbers. Efforts to control this group have been discouraging to both the public and our profession. A deep distrust of organized medicine has ensued, and productive research has been enormously handicapped. But one encouraging fact remains: Chemotherapy for cancer in man, as limited, difficult, and noncurative as it is, offers a ray of hope. We can now produce cancer in lower animals by transplantation, and can cure them! Not enough attention has been given to the cancer cell itself as a cancer-producing agent. We need to look upon it in a way similar to our attitude toward disease-producing germs. Now we can inhibit the active growth of cancer in man temporarily, and destroy it completely in experimental animals. Our view may well parallel that of the disease-producing parasitic micro-organisms. We need to turn to the postulates of Koch once more. These are the following: 1. The organism must be regularly present in all affected tissues; (2) it must be cultivatable in pure form; (3) one must be able to reproduce the disease by inoculation of the organism. A fourth postulate may now be set up: the resistance acquired by cells against our therapeutic agents. Localized therapy is not enough; we must be

able to deal effectively with disseminated cancer, that is, cancer which has spread throughout the body. We must achieve selective chemical destruction. Now we are able to keep alive neoplastic cells outside the body in pure culture for months! Reimplantation shows that the tissue grows well, and when excised was not distinguishable from the original cultured cells or from the original implant. The neoplastic cell has many attributes found in other invading enemies, especially bacteria. We may well make analogous experiments from which analogous conclusions can be drawn. For over 50 years we have known some of the effects of certain hormones in inhibiting growth of breast cancer. Huggins, in 1941, applied this principle to prostatic cancer by removing the testicular androgenic stimulus to prostatic cancer.

A second advance in the chemotherapy of cancer came about during World War II in the use of nitrogen mustards, which are toxic to cancer cells. However, there are some disadvantageous by-products on normal cells in the mouth, intestinal mucosa, and hematopoietic structures. Another observation: Cell division and propagation cannot go on save in the presence of folic acid. Therefore, if we could prevent folic acid from entering cancer cells, might we not starve these anarchists? Again, modified "adenines" are capable of blocking certain cells from reproducing.

An experimental result is well worth quoting: "In our laboratories, in experiments by Drs. Stock, Suguiara, Philips, Clarke, and their associates in more than 2,000 experimental animals bearing six different types of transplantable cancer, over 70 percent of the neoplasms have been completely destroyed with cure of the hosts." Failure of the implanted tumor to grow in the treated animals when transplanted back into the bodies of untreated animals shows that somehow inhibition was highly effective. Applying all this to man may not bring the expected and hoped for results, but it is highly encouraging to know that in nonhuman animals cancer can be cured! What a booster for vivisection!

Some satisfactory experiments with an agent known as Purinethiol in transient control of leukemia are of great interest. A symposium from 25 clinics showed remarkable uniformity of results. It was used in addition to cortisone and chemicals having antifolic activity, with a survival of children for over one year, an increase of from 29%-52%, enabling these children to lead fairly normal lives during the remission periods. Recurrence, however, is likely, it is sad to state.

In summary, transplantable cancer can be completely destroyed in experimental animals. On occasion, certain cancers can be restrained in humans! Rhoads advances the hope that chemicals yet to be put together may eventually restrain or destroy cancer in humans. This hope will reecho around the world, since it is based on scientific studies of such high degree of excellence.

I. W. VOORHEES, New York.

TREATMENT OF BRAIN ABSCESS. ALBERT W. COOK, WILLIAM H. DRUCKEMILLER, and HOWARD FREEDMAN, New York J. Med. **54**:3366 (Dec. 15) 1954.

This paper was read at the Annual Meeting of the Medical Society, State of New York, on May 14, 1954. It is based on studies at the University of the State of New York, Department of Neurosurgery, in the College of Medicine.

By definition, "An abscess is a circumscribed area of suppuration in which tissue necrosis is present." In the brain, there is widespread invasion, focal suppuration, or spontaneous resolution. Treatment is by antibiotics or surgery or both. The antibiotics reduce infection and allow time to build up host resistance and often obviate lethal meningitis. No one method is paramount, but a "burr hole" may be a valuable adjunct for sufficient drainage before surgery of the brain itself. At least, acute symptoms are relieved, and there is time for planning the further management. In general, symptoms are headache, nausea and vomiting, papilledema, hemianopsia, and hemiparesis (not always present). Simple surgical procedures (drainage) may be sufficient, but excision of the abscess may be called for. Much reliance can be put on chemotherapy as a valuable aid to convalescence. But it must be remembered that brain necrosis may require complete removal of all necrotic tissue (if drainage is insufficient).

ABSTRACTOR'S COMMENT: Four cases are cited in support of the authors' method of management. This is insufficient evidence, as the case histories are too sketchy. Few surgeons of experience are willing to concede that "a simple trephine opening combined with massive doses of antibiotic drugs (will result) in most instances in subsidence of symptoms and eventual recovery with minimal sequelae."

I. W. VOORHEES, New York.

ACUTE TRANSITORY ATAXIA IN YOUNG CHILDREN. P. DENYS and L. GERIN, Acta pædiat. belg. **7**:99, 1953.

The authors describe the little-known syndrome of disturbances of the equilibrium in small children occurring in conjunction with or after acute viral diseases, together with seemingly banal upper respiratory infections or without any accompanying symptoms, as a primary disease, possibly caused by a poliomyelitis virus. The symptoms occur suddenly, last from a few days to two weeks, and disappear without sequelae. The few existing literature references are discussed and 14 cases presented.

The symptoms in the reported cases were either purely cerebellar or cerebellovestibular. In five cases they occurred in the course of upper respiratory infections; in one, after chickenpox, and in one, after probable viral pneumonia. The last five cases occurred in short succession during an epidemic of poliomyelitis.

GLASER, Jerusalem [A. M. A. AM. J. DIS. CHILD.].

CLINICAL STUDY OF CEREBRAL ABSCESS IN INFANTS. C. BEECKMANS, Acta pædiat. belg. **7**:263, 1953.

The case of a 9-month-old infant who was admitted because of physical and psychomotor retardation is reported. The head appeared large and asymmetrical; the fontanel was tense, and there was intermittent strabismus. The electroencephalogram showed focal signs in the left temporo-occipital region. A pneumoencephalogram showed signs of a space-occupying lesion on the left side. A staphylococcic abscess in the left temporal region was opened, drained, and treated by daily injections of penicillin and streptomycin through the drain. The child recovered, and electroencephalographic findings returned to normal.

The child's past history revealed a maternal breast infection when the infant was one month of age and pyelitis 14 days before admission.

The author considers local trauma, direct propagation of infection, and metastatic infection as etiologic agents. The presenting symptoms were intracranial hypertension, signs of infection, and neurological signs permitting localization with the aid of electroencephalogram, pneumoencephalogram, lumbar puncture, and, finally, puncture of the abscess.

GLASER, Jerusalem [A. M. A. AM. J. DIS. CHILD.].

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NATIONAL

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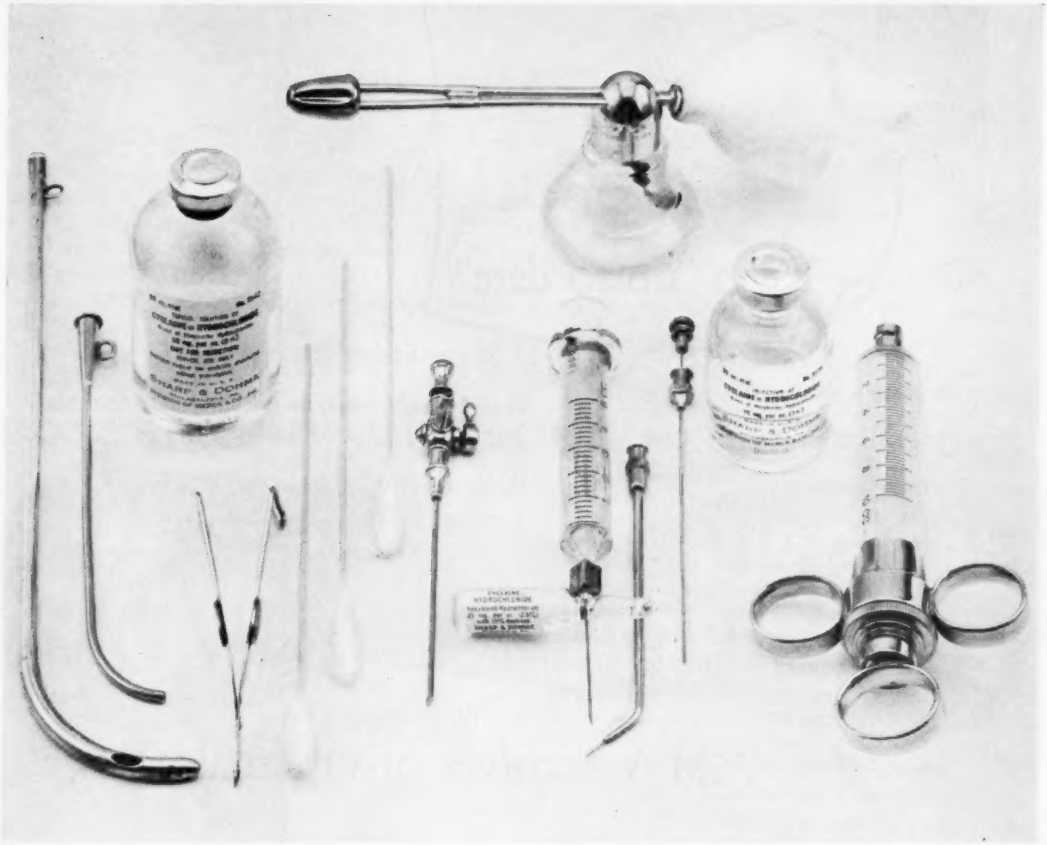

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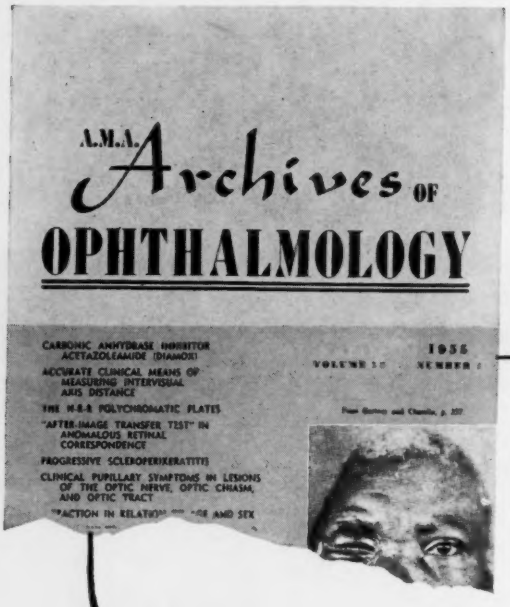
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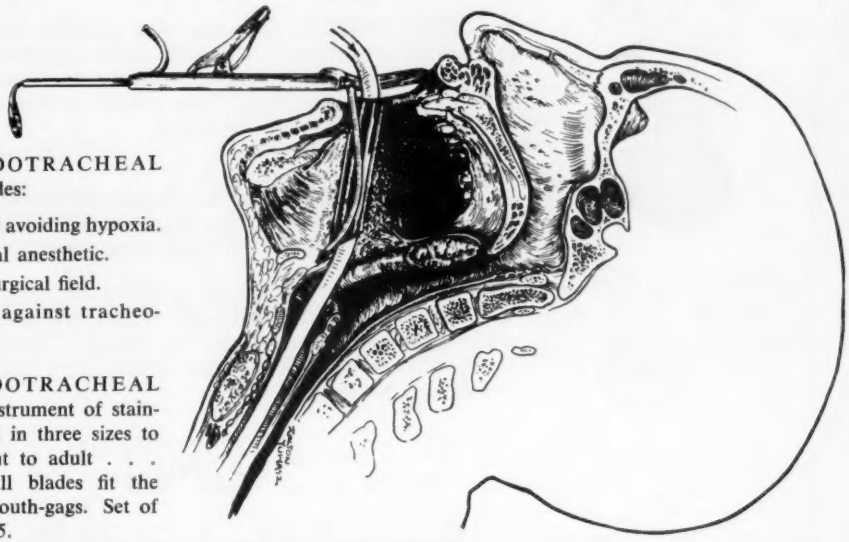

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*Barton, R. T.: Instrument for endotracheal anesthesia during oro-pharyngeal surgery. J. A. M. A. 153:1017 (Nov. 14) 1953.

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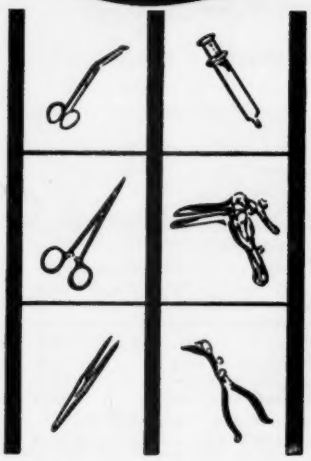

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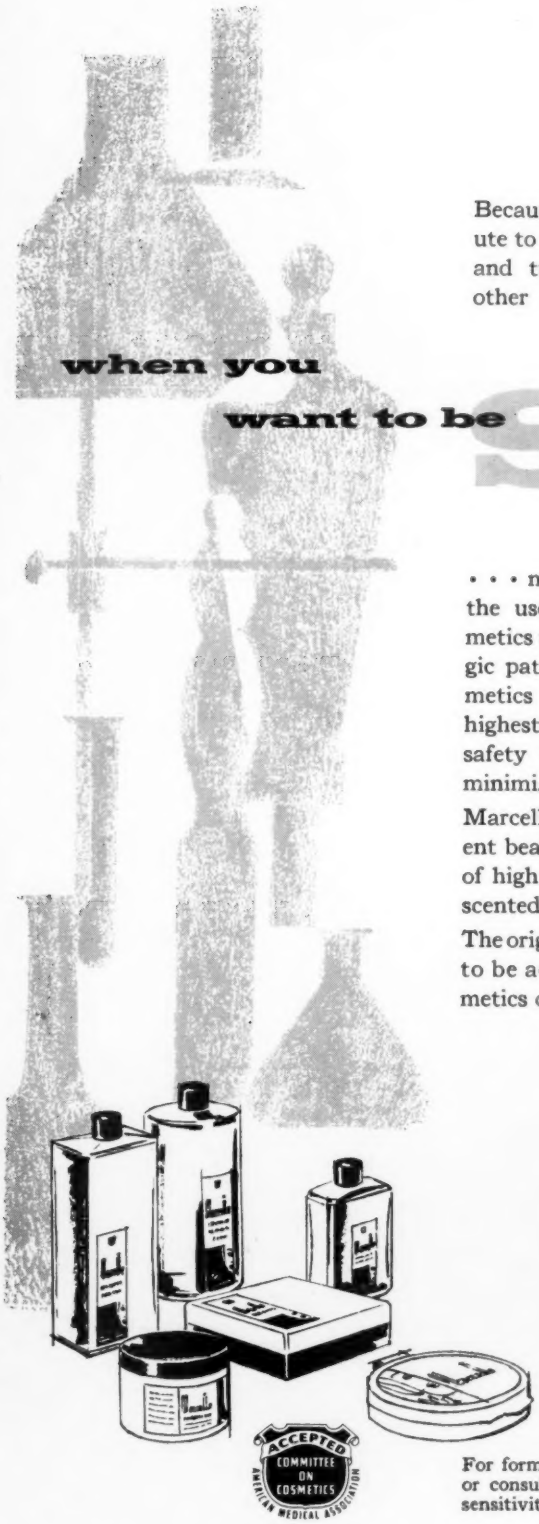

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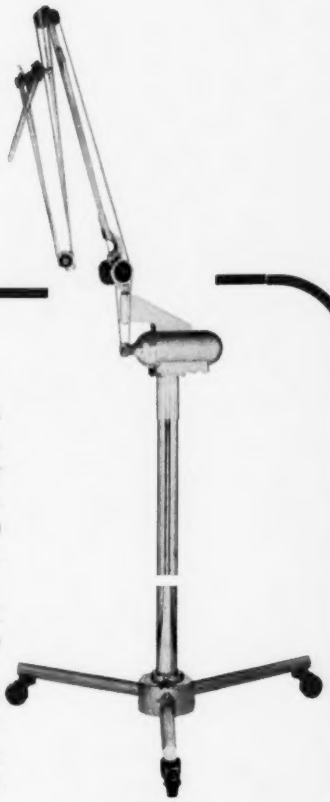

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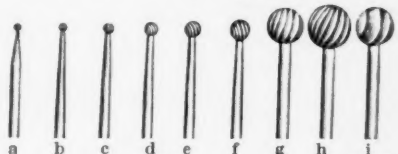

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